Health risks of exposure to depleted uranium

An overview

Gezondheidsraad **Vice-president**
Health Council of the Netherlands

To the Minister of Housing, Spatial Planning and Environment

Subject :
Your reference :
Our reference : U 1027/WP/MK/678
Enclosure(s) :
Date : 16 May, 2001

I hereby present you with the advisory report 'Health Risks of Exposure to Depleted Uranium'. The report is the work of a specially established committee of the Health Council. During its deliberations, the committee drew upon advice obtained from the Standing Committee on Environmental Factors and Health and the Standing Committee on Radiological Protection. I have today also submitted copies of this report to the Minister of Defence and the Minister of Health, Welfare and Sport.

Further reports on depleted uranium have very recently been published by both, the global and European arms of the World Health Organization, and by the United Nations Environmental Programme These reports were not available when the committee was considering the issue, but on preliminary examination they do not appear to contradict the committee's findings.

Yours sincerely,

(signed)
Prof. dr JA Knottnerus

P.O. Box 16052
NL-2500 BB The Hague
Telephone +31 (70) 340
Telefax +31 (70) 340 75 23
E-mail:

Visiting Address
Parnassusplein 5
NL-2511 BX The Hague
The Netherlands

Health risks of exposure to depleted uranium

An overview

Report of a committee of the Health Council of the Netherlands

to:

the Minister of Housing, Spatial Planning and the Environment

the Minister of Defence

the Minister of Health, Welfare and Sport

Nr 2001/13E, The Hague, 16 May 2001.

The Health Council of the Netherlands, established in 1902, is an independent scientific advisory body. Its remit is "to advise the government and Parliament on the current level of knowledge with respect to public health issues..." (Section 21, Health Act).

The Health Council receives most requests for advice from the Ministers of Health, Welfare & Sport, Housing, Spatial Planning & the Environment, Social Affairs & Employment, and Agriculture, Nature Preservation & Fisheries. The Council can publish advisory reports on its own initiative. It usually does this in order to ask attention for developments or trends that are thought to be relevant to government policy.

Most Health Council reports are prepared by multidisciplinary committees of Dutch or, sometimes, foreign experts, appointed in a personal capacity. The reports are available to the public.

Preferred citation:
Health Council of the Netherlands: Health risks of exposure to depleted uranium. The Hague: Health Council of the Netherlands, 2001; publication no. 2001/13E. Revised version 5 February 2002

ISBN: 90-5549-387-2

Contents

Executive summary

Health Council of the Netherlands: Health risks of exposure to depleted uranium The Hague: Health Council of the Netherlands, 2001; publication no. 2001/13

Not surprisingly, the use of ammunition containing depleted uranium (DU) in Kosovo and elsewhere in the Balkans has provoked disquiet in Europe. In the Netherlands, concern over the release of this material had already been aroused previously following the crash of the El-Al airliner in the Bijlmermeer district of Amsterdam in 1992. It was against this background that the President of the Health Council decided to set up a Committee charged with the task of reviewing the health risks of exposure to DU and the preventive measures required for individuals present in areas where DU has been released into the environment. The present advisory report provides this review.

Uranium and depleted uranium

In its pure form, uranium (U) is a heavy, silver-coloured, radioactive metal. It is ubiquitous in nature in its natural isotopic form, together with its radioactive decay products. These decay products are removed during the extraction of uranium from ore. Natural uranium consists principally of the isotope U-238 and, to a minor extent, the isotopes U-234 and U-235 .

Depleted uranium is recovered as a by-product of natural uranium during the enrichment of uranium for use in nuclear power stations. DU is characterised by a reduction to 0.2% of the percentage of the isotope U-235. The radioactivity per unit of mass (activity concentration) of DU is lower than that of natural uranium (the

respective levels being 14.8 and 25.4 kilobecquerels per gram). DU behaves chemically (and with that also toxicologically) identical to uranium in its natural isotopic form.

In residues of DU from Kosovo a small amount of U-236 (0.0028%) has also been detected. The contribution made by this isotope to the total activity concentration is so minute that it does not influence the radiological properties of DU. From information regarding the possible contamination of this DU with transuranium elements (including plutonium) and fission products we can surmise that these impurities do not play any significant role in the assessment of the risk of exposure to DU.

Uranium in the living environment

Uranium occurs naturally in the environment and therefore also in the human body. In the Netherlands, the concentration of uranium in the soil varies between 0.4 and 8 milligrams per kg of dry earth. Intake of uranium by humans principally occurs via the diet. Most of the orally ingested uranium is eliminated from the body in the excreta (principally in the faeces), but some accumulates in body tissues (mainly in bone).

DU enters the living environment via specific events, like a fire of objects containing DU (example: Bijlmer disaster) and military use of DU (example: Gulf War). Following such events DU fragments might be found in the areas concerned and uranium dust, usually in the form of the slightly soluble oxides, might have spread around in the surroundings of the event. That can cause exposure of the population, in particular via consumption of foodstuffs which have been grown on the contaminated soil and via inhalation of resuspended dust containing DU. Relief workers and military personnel entering these areas might be exposed via inhalation of dust and by radiation from DU fragments.

Exposure in the framework of other applications of DU will be limited to occupational exposure. These situations are not treated in the present advisory report. Exposure of soldiers during military actions is entirely left out of consideration. The DU exposure mentioned above comes on top of the exposure to natural uranium in the living environment.

Uranium in the body

Absorption, distribution and excretion in the human body are highly dependent on the chemical form of the uranium and on the manner in which it is entering the body. Thus slightly soluble compounds such as uranium dioxide are only slowly eliminated from the lungs and will therefore only burden other organs in minute quantities. Ingestion of slightly soluble compounds results in little or no contamination of the body, since there

is very limited absorption through the intestinal wall, by far the greater part being excreted in the faeces. Soluble compounds, on the other hand, are able to enter partly the blood circulation via the lungs or the intestinal wall and they then accumulate in organs (especially in bone). However, the great majority is excreted relatively quickly in the urine.

Concentrations of 1 to 3 micrograms of uranium per kg of wet tissue are typically detected in organs. Typical values for excretion range from 0.05 to 0.5 micrograms per day in urine and around 1.5 micrograms per day in faeces.

Health effects of exposure

When assessing the health effects of exposure to natural uranium and DU, it is necessary to consider both the radioactivity of the material and its chemical toxic effect. Based on existing knowledge of the radiological properties of uranium, it would appear that radioactive contamination of the lungs is the principal health effect to be considered in connection with exposure to slightly soluble uranium compounds in the atmosphere. In this context should be mentioned that the dose arising from exposure to DU is much smaller than from exposure to natural uranium per unit of mass. For soluble compounds, the chemical toxic effect in the kidneys is the primary consideration. The toxicological effects are to some extent concordant with those of other heavy metals.

A substantial amount of work has been done with uranium since the mid twentieth century. Research involving large groups of workers in the uranium industry has produced valuable data about the risks of exposure to uranium, but it also displays the frequently unavoidable shortcomings: namely, substandard information about the actual exposure of the workers, substandard or non-existent information on exposure to other possibly harmful agents and unsatisfactory data on disruptive variables such as smoking habits.

The epidemiological research has not produced any clear evidence that exposure to uranium leads to health impairment. According to the literature, the additional cases of lung cancer among workers in uranium mines are attributable to the inhalation of the radioactive decay products of radon, which is found in elevated concentrations in and around mines. Military personnel who took part in the Gulf War exhibit more health complaints than others do. The extensive investigations conducted among these veterans have produced no evidence that exposure to DU is a causative factor in these complaints.

The Committee does not, therefore, anticipate that exposure to DU in the situations described above, also given the possible extent of the exposure, will result in

a demonstrable increased risk of diseases and symptoms among exposed individuals as a result of a radiological or chemical toxic effect exerted by this substance.

Cancer

In view of the fact that DU emits ionising radiation in the form of alpha particles, the induction of cancer, in principle, needs to be taken into account in relation to individuals exhibiting internal contamination with DU. In case of inhalation of slightly soluble DU compounds, attention will in particular need to be focused on the lungs.

The radiation dose caused by incidental exposure to DU in the outlined scenarios is in the most conceivable cases limited compared with the radiation dose received during a lifetime of natural uranium. Since at the usual levels of exposure to natural uranium a contribution to the induction of cancer in the population cannot be shown it can be concluded that the same is true for exposure to DU in the outlined scenarios. This general conclusion is also valid for the appearance of lung cancer and for the appearance of leukaemia after the inhalation of dust containing slightly soluble uranium compounds. The radiation dose elicited in the bone marrow and with that the theoretical probability for cancer induction by slightly soluble compounds is three orders of magnitude smaller than that elicited in the lungs.

Renal damage

For soluble compounds, the risk posed by exposure to DU is principally of a chemical toxic nature. In the case of increasing exposure, abnormalities will first of all appear in the kidneys. Exposure to small amounts (milligrams) of uranium over short periods will therefore result in changes in the kidneys which lead to acute, usually reversible, renal impairment. No such dose-dependency has been observed, however, in the frequency of chronic renal disorders among population groups who are chronically exposed to –in general totally less than 1 milligram per year– natural uranium. Nor have studies involving workers in the uranium industry and ex-military personnel (including the group with shrapnel in the body) to date produced any evidence that uranium can cause renal impairment. Thus the present body of scientific data tends to suggest an absence of irreparable renal damage as a result of the intake of DU in the exposure scenarios considered.

Prevention

DU is, just like several other heavy metals, classified as a hazardous substance. It is, after all, evident from the foregoing findings that the risks associated with exposure to DU for the exposure scenarios outlined here are very limited. The fundamental principle adopted in the fields of industrial and environmental hygiene dictates that unnecessary exposure to a hazardous substance must be avoided. According to the so-called ALARA principle (As Low As is Reasonably Achievable), exposure must be avoided as far as is reasonably possible. 'Reasonably' implies that the efforts made must be commensurate with the achievable degree of risk reduction – i.e. the achievable reduction in exposure.

As far as possible exposure to DU in contaminated areas is concerned (for example in the vicinity of a fire in which DU has been released or in an area where military actions involving the use of DU ammunition have occurred), the Committee considers the strategy for protection laid down in the rules and regulations governing radiation protection to be adequate, both as regards limiting radiological and chemical toxic risks. This means that the first priority is to determine the nature and the extent of the contamination. Has contamination actually occurred? After that questions should be raised like: If so, how extensive is it and which compounds are involved? Are there fragments of DU in the area? Is there a possibility that windblown DU compounds could be inhaled? And so on. Based on the answers to these questions, one can determine whether it is necessary to impose limitations on access to, and use of, the contaminated area, and whether or not individuals who need to enter the area in a professional capacity (relief workers, for example) should be regarded as radiological workers. This will presumably only apply be necessary in exceptional cases.

The answers on the foregoing questions are not only of importance for experts and authorities but also for the population in the vicinity of the place where DU has been liberated and for persons who for occupational reasons have to stay there. Open communication can prevent unnecessary anxiety. For this the Committee refers to an other recently published advisory report of the Health Council 'Local environmental health concerns'.

Chapter 1

Introduction

1.1 Background

Exposure to uranium, in particular depleted uranium (DU), has been the subject of much debate in the Netherlands since an El Al aeroplane crashed in the Bijlmermeer in 1992. Public interest in the issue was further stimulated when it became known that during the 1999 Kosovo crisis, NATO had fired roughly 31 000 DU-containing shells in areas where Dutch KFOR troops and aid workers may have been present.[1] DU-containing ammunition may previously have been used during the Bosnian conflict as well.[2]

A working group of UNEP (the United Nations Environmental Program) has looked into the repercussions of the Kosovo conflict for human health and the environment. In 1999, this working group concluded – pending the outcome of follow-up research scheduled for publication in 2001 – that, despite the availability of information from NATO, there was not enough data for reliable assessment of the likely environmental and public health implications of using DU ammunition in Kosovo.[3] A follow-up report based on inspections and tests conducted in the former war zone in November 2000 was published as planned by UNEP in 2001.[4]

Other organisations that have considered the health effects of exposure to DU include the World Health Organisation (WHO) and the UK's Royal Society. In addition, the use of DU during the Gulf War has been studied extensively. In 1999,

RAND's National Defense Research Institute in the USA published a survey of available data on the effects of DU on human health.[5] More recently, the American Institute of Medicine has produced a report on health issues associated with the Gulf War; as well as looking at DU, this report considered other possible causes of health problems experienced by veterans of the conflict.[6]

1.2 Commission, composition of the committee and approach

Against the background outlined above and following official contact between the Council's Secretariat and the Ministries of Defence and Housing, Spatial Planning and the Environment, the President of the Health Council decided in May 2000 to establish a committee to undertake a survey of:

- the health risks of exposure to depleted uranium and
- appropriate precautions for people working or otherwise present in areas where depleted uranium has been released into the environment.

The members of the committee are listed in annex A.

This document contains the findings of the committee's survey. Distinction has been made between exposure to DU in the general environment – through the intake of food or water, for example – and occupational exposure involving workers and military personnel. Where the armed forces are concerned, the committee has confined itself to exposure in the course of peacetime operations and has not considered exposure during hostilities

Since the chemical toxicity of DU is consistent with that of uranium with a natural isotopic composition, the committee felt it should also take account of naturally occurring uranium present in the biosphere and the food chain ('natural uranium'). Having been used in the nuclear industry since 1945, a great deal is known about natural uranium. 'Enriched uranium' – produced, for example, by Urenco in Almelo for use in nuclear power plants such as that at Borssele – isoutside the scope of this report.

The primary sources of information consulted by the committee were survey reports, particularly those referred to above concerning the consequences of participation in the Gulf War for the health of (former) American military personnel, and a toxicological profile produced by the Agency of Toxic Substances and Disease Registry in the USA.[5,6,7] Where it was considered necessary, the committee also referred back to the original publications upon which these surveys were based. Recent

publications on uranium and depleted uranium were also obtained from the *Medline* database and reviewed by the committee.[a]

1.3 Structure of this report

The body of this report starts by summarising the properties and applications of uranium (chapter 2), the natural occurrence of uranium (chapter 3) and exposure to uranium (chapter 4). Chapters 5 and 6 are devoted to the toxicity of uranium and uranium compounds. The basis on which recommended exposure limits are calculated is then considered in chapter 7. Finally, the committee's conclusions regarding the health risks of exposure to depleted uranium and its recommendations regarding the precautions that should be taken to limit exposure are presented in chapter 8.

[a] In addition to the sources referred to in this report, information about uranium is constantly being made available via the Internet. Sites from which toxicity data is available include:

- toxnet.nlm.nih.gov
- databases such as Toxline, HSDB, etc.

General information on uranium can also be found at:

- www.uic.com.au/index.htm
- www.uilondon.org/
- www.nndc.bnl.gov/

Chapter 2

Properties and applications of uranium

2.1 Introduction

Pure uranium (chemical symbol: U) is a silver-coloured, heavy, radioactive metal. The element occurs naturally and is present generally in the environment. First identified by Klaproth in 1789, uranium was named after the newly discovered planet Uranus. Uranium ores may contain anything from a few hundredths to a few dozen percent uranium by weight. The natural isotopic composition by weight is 99.2739 per cent U-238, 0.7204 per cent U-235 and 0.0057 per cent U-234.[a] The activity concentration (radioactivity per unit weight) of natural uranium is roughly 25 Bq per milligram.[b] DU, which contains less U-235 than natural uranium, is formed as a by-product of uranium enrichment. The typical isotopic composition of DU is 99.8 per cent U-238, 0.2 per cent U-235 and 0.001 per cent U-234. DU with this composition has an activity concentration of 14.8 Bq/mg.

DU is sometimes produced using partly recycled uranium from nuclear reprocessing plants. Such DU contains a small amount of U-236, an isotope that is formed in nuclear reactors during the fission process. The AC Laboratory in Spiez, Switzerland, has established that an ammunition sample collected by the UNEP in Kosovo contained 0.0028 per cent U-236.[8] Recycled DU can also contain other

[a] See glossary.
[b] See glossary.

transuranic elements (americium, neptunium, plutonium) or fission products from spentnuclear fuel. Although this was indeed the case with the sample tested in Switzerland, the test results indicated that the concentrations in which these substances were present were not significant either in radiological terms or in terms of chemical toxicity.[9]

Depleted uranium has various industrial, research and military applications. Metallic DU is used for mass balance weights in aircraft[a], for anti-tank ammunition, for heavy tank armour, as a detector material in high-energy physics and as shielding material for strong radioactive sources, for example. In all these cases, only depleted uranium is used. The uranium used for nuclear reactor fuel almost always has a substantially higher U-235 concentration than natural uranium; it is not therefore appropriate to consider this material in the present context.

2.2 Physical and chemical properties

The physical properties of natural, elemental uranium are summarised in table 2.1. Uranium is a reactive metal with a high density: it is 1.7 times as dense as lead and nearly as dense as tungsten. It will form binary alloys with most other metals, while its mechanical properties make it easy to process. It is also slightly paramagnetic. In nature, uranium does not occur in its metallic form, but as a component of minerals such as carnotite, uraninite and pitch blend. Like aluminium powder and magnesium powder, uranium powder is pyrophoric (self-igniting). At room temperature, it is liable to ignite spontaneously in the presence of air, oxygen or water. At temperatures of 200 to 400°C, uranium powder can also ignite in an atmosphere of carbon dioxide or nitrogen.[7] When exposed to the atmosphere, pure metallic uranium rapidly oxidises; a thin layer of uranium oxide (UO_2) is formed at the surface, preventing further oxidation of the material below.

Table 2-1 Some of the properties of natural uranium.[10]

Atomic weight	238.03 g/mol
Density (25 °C)	19.214 g/cm³ (X-ray diffraction)*
	19.05±0.02 g/cm³ (experimental)
Boiling point	3818 °C
Melting point	1132.3 °C

* Calculated from crystal structure measured with x-ray diffraction. Standard reference value.

[a] Tungsten is nowadays preferred for this application.

Uranium in compounds is found in five oxidation states: +2, +3, +4, +5 and +6. However, only the +4- and +6 states are sufficiently stable to be of any practical significance. Quadrivalent (+4) uranium forms oxides, hydroxides, hydrated fluorides and phosphate compounds that exhibit limited solubility. Hexavalent (+6) uranium is the most stable form, most commonly occurring in the oxide U_3O_8.[a] The uranyl ion ($[UO_2]^{++}$) fluoresces under the influence of ultraviolet light (see also 2.4). Uranium compounds vary considerably in terms of the solubility in water: solids, such as uranium dioxide (UO_2), uranium trioxide (UO_3) and tri-uranium octaoxide (U_3O_8) are not soluble in water, although they are soluble in sulphuric acid and nitric acid. Uranium tetrachloride (UCl_4), uranyl fluoride (UO_2F_2) and hydrated uranyl acetate ($UO_2(CH_3COO)_2.2H_2O$) dissolve easily in water, however. Uranium oxides are slow to dissolve in bodily fluids. The solubility of uranium compounds is very important in relation to their impact on human health. The International Commission on Radiological Protection (ICRP) distinguishes between three classes of uranium compound on the basis of solubility in bodily fluids: F, M and S (fast, medium and slow), reflecting the rate of elimination from the lungs.[11] Table 2-2 contains a summary of the properties of the most common uranium compounds. Uranium shares many chemical properties with other heavy metals. Heavy metal compounds are generally highly reactive.

Table 2-2 Physical and chemical properties of selected uranium compounds.[12]

Compounds (CAS-numbers)	Molecular formula	Mol. weight (g/mol)	Appearance	Solubility in water	Absorption type ICRP
Uranium tetrafluoride (10049-14-6)	UF_4	314	green triclinic needles	slightly soluble	M
Uranium hexafluoride (7783-81-5)	UF_6	352	colourless crystalline, deliquescent	decomposes	F
Uranium tetrachloride (10026-10-5)	UCl_4	380	dark green metallic	soluble	M
Uranium tetraiodide (13470-22-9)	UI_4	746	black needles	soluble	M
Uranium dioxide (1344-57-6)	UO_2	270	brown/black powder	insoluble	S
Uranyl oxide (1344-58-7)	UO_3	286	red/brown powder	insoluble	M
Triuranium octaoxide (1344-59-8)	U_3O_8	842	olivegreen - black	insoluble	S
Uranyl acetate (541-09-3)	$UO_2(C_2H_3O_2)_2.2H_2O$	422	yellow crystal powder	soluble	F
Uranyl nitrate (36478-76-9)	$UO_2(NO_3)_2.6H_2O$	502	yellow crystals	soluble	F
Uranyl phosphate (18433-48-2)	$UO_2HPO_4.4H_2O$	438	yellow microcryst powder	insoluble	S

[a] This compound contains pentavalent and hexavalent uranium.

2.3 Radiological properties

Becquerel first established that uranium was radioactive in 1896. The element has twenty-two known isotopes, of which only three occur naturally: U-234, U-235 and U-238.

Table 2-3: Radiological properties of natural and depleted uranium (activity of decay products not included).[13]

Isotope	Half-Life (million years)	Alpha Particle Energy (MeV)(%)	Natural uranium		Depleted uranium	
			Isotopic (%)	Activity (Bq/mg U)	Isotopic (%)	Activity (Bq/mg U)
U-238	4468	4.147 (23) 4.196 (77)	99.2745	12.40	99.8000	12.40
U-234	0.2450	4.724 (28) 4.776 (72)	0.0055	12.40	0.0010	2.26
U-235	703.7	4.364 (11) 4.395 (55)	0.7200	0.60	0.2000	0.16
total				25.40		14.80
U-236	24	4.5				

The radiological properties of natural and depleted uranium are summarised in table 2-3. It will be apparent from the data presented that the level of activity exhibited by a given weight of pure depleted uranium is only about half of that exhibited by a similar amount of pure natural uranium. All isotopes of uranium are radioactive and gradually decay in a number of stages to form stable lead isotopes. The decay schemes for U-235 and U-238 are given in annex B. Radioactive decay results in the release of ionising radiation, in the form of alpha particles, beta particles and gamma radiation. Alpha and beta particle energy data is presented in table 2-4

In nature – and therefore in ores – uranium is always found in the presence of the decay products listed in annex B. When uranium is extracted from an ore, it is separated from these radioactive decay products, with the result that processed uranium initially contains only the isotopes U-234, U-235 and U-238. After several months, however, the short-lived isotopes – thorium-234 and protactinium-234 (from U-238) and thorium-231 (from U-235) (see annex B) – return to their previous levels. This is why, a while after refinement, uranium emits beta particles as well as alpha particles. Very little gamma radiation is emitted from refined uranium, however.

It is important to note that the activity concentration of uranium, as it occurs naturally in ores or in the ground, is very different from that of refined uranium,

whether its composition is 'natural' or depleted. As a result of the radioactive decay process, the activity concentration of depleted and refined natural uranium increases from the levels given in table 2-3 to 39.42 and 50.23 Bq per milligram of uranium, respectively, within a few months.[14] Data on the radioactivity of various forms of uranium is presented in table 2-5

Table 2-4 Energy and range of alpha and beta particles in air and tissue.

Type of radiation	Energy (MeV)	Range	
		Air (m)	Tissue (µm)
Alpha particles U-238/235/234	4.15-4.78	0.026-0.03	35
Beta particles Pa-234*	2.3 (maximaal)	7.6	11000
* protactinium-234 (Pa-234) is a decay product of uranium-238			

The risk from exposure to ionising radiation is associated with the transfer of energy to biological material. Alpha particles cannot penetrate more that 35 micrometres (µm) into (soft) biological tissue (see table 2-4. In the event of external exposure, therefore, almost all the energy from alpha particles is absorbed by the dead outer layers of skin. However, alpha particles can play a more significant role where internal exposure is concerned, damaging cell structures within their range (measured from the point of deposition). If they have sufficient energy, beta particles can penetrate the skin far enough to reach sensitive cells and therefore present a potential hazard even where external irradiation is concerned. Nevertheless, it is again internal exposure that brings the greatest risk. Gamma radiation is highly penetrative and consequently forms a hazard in the event of internal or external exposure.

As the data in table 2-3 shows, DU (and natural uranium) has a comparatively low activity concentration compared with other radioactive substances. Hence, contact with metallic uranium entails relatively little risk from external irradiation by beta particles or gamma radiation (see also chapter 4). By way of comparison, it is worth noting that the activity concentration of potassium-40 – a naturally occurring isotope that accounts for 0.1 per cent of the potassium in the human body – is 250 Bq/mg. The activity concentration of radium-226 – a decay product of uranium-238 (see annex B) – is 3.7×10^7 Bq/mg.

Table 2-5 Radioactivity of various forms of uranium

Form	Activity Bq/mg
Natural uranium (with all decay products)	180.2
Natural uranium immediately after processing	25.50
Natural, processed uranium after a few months	50.23
Depleted uranium immediately after production	14.80
Depleted uranium a few months after production	39.42

Table 2-3 also gives data on U-236, traces of which are sometimes found in depleted uranium made using recycled uranium (see 2.1). Trace quantities of U-236 can also occur in unprocessed natural uranium, since the isotope may be formed in natural nuclear reactions, albeit in smaller quantities than those produced in a nuclear reactor. The reaction by which U-236 is formed entails the gradual capture of neutrons by U-235. In a manmade reactor, a plentiful supply of neutrons is available from the fission of uranium nuclei. In nature, the reaction is enabled by the release of neutrons from the spontaneous fission of uranium and by the availability of neutrons of cosmic origin.

2.4 Testing for uranium

Both chemical and radiological techniques are used to determine uranium concentrations. Soil samples are typically analysed by gamma-spectrometry using germanium detectors – a technique which is capable of detecting a uranium activity level of 20 Bq or more (1.4 mg) in a 200-gram sample. For the measurement of uranium levels in urine, fluorimetry is generally the preferred option. Using this technique, the minimum detectable concentration is 1 µg/l[a]. Lower concentrations (0.001-0.06 µg/l) can be detected by means of laser fluorimetry.[15] The first-choice method for the analysis of 'natural' samples is nowadays a special form of mass spectroscopy (HR-ICP-MS), which allows the detection of less than 0.0001 µg/l.[16] With HR-ICP-MS, it is also possible to distinguish between natural and depleted uranium. Another very sensitive technique is neutron activation analysis, which can also be used to determine the uranium concentration in the urine of people from an area characterised by 'normal' background concentrations of environmental uranium.[17] The sensitivity of this technique is sufficient to detect 0.0002 µg U-238.

Alpha spectrometry is also widely used to measure uranium concentrations. This technique has the advantage of showing what the isotopic composition is, so that distinction can be made between depleted and natural uranium. With alpha spectrometry, the detection limit is 0.1 mBq for a twenty-four-hour urine sample (0.007 µg DU) or 1 mBq (0.07 µg DU) for a twenty-four-hour faeces sample.[18]

Even long after a single brief period of exposure, it has proved possible to detect an increase in uranium excretion levels in cases where accurate data on pre-exposure concentrations was available. Using techniques of this kind, overall exposure figures can also be calculated.

[a] micrograms per litre

2.5 Military applications

DU has been in military use since 1970. It is favoured because of its high specific mass, hardness and low-temperature combustion properties. One of the main military applications is the manufacture of anti-tank ammunition: shells that literally burn their way through the armour of a tank.

The temperature at which uranium ignites depends on the ratio between the surface area and volume of the uranium-containing material. The combustion of DU shells results in the release of aerosol particles. High combustion temperatures result in the production of oxides: mainly U_3O_8, but also UO_2 and UO_3. Weathering leads to the gradual subsequent oxidation of any uranium particles not oxidised at the time of combustion.

DU is also used in heavy tank armour, primarily on account of its hardness and high density.

2.6 Other applications

As indicated in section 2.1, DU is used in the aviation industry to make mass balance weights and in high-energy physics as an experimental detector material. The ZEUS experimentat Germany's DESY research institute incorporates detector plates that contain many tons of DU, for example; certain Dutch institutes have been actively involved in the construction and use of this rig.

Bijlmer disaster

The use of DU in mass balance weights came to the attention of the general public following an aviation disaster on 4 October 1992, when an El Al Boeing crashed into the Bijlmermeer.[19] The cargo plane involved was carrying twenty-four pieces of DU as mass balance weights. These weights were each between six and thirty kilos, with a combined mass of 282 kg at the time of the accident. Only 130 kg of DU was found at the crash site, the fate of the remaining 152 kg is still not known. Some may have been taken away with the polluted soil subsequently removed from the site. It is also possible that the uranium was partially oxidised in the burning wreckage and dispersed into the environment. This scenario has been thoroughly investigated by Uijt de Haag *et al.*[20] Uranium oxide formed under such circumstances would not be readily soluble, so the health risks associated with intake would be determined mainly by the oxide's radiological properties (see below). Calculations indicate that, if this is what happened

to the DU, rescue workers would have received a radiation dose[a] of less than 1 microsievert (µSv)[b].

[a] Committed effective dose equivalent; see glossary.

[b] A radiation dose is expressed in terms of effective dose in the case of averaging over the whole body, while the average dose to a particular organ is expressed as an equivalent organ dose. In these quantities the effectiveness of the radiation to cause biological damage (in particular cancer) is taken into account.. See also the glossary.

Chapter **3**

Naturally occurring uranium

Uranium occurs naturally in the environment and is therefore normally present in the human body. Natural uranium includes the isotopes U-238, U-235 and U-234 (see table 2-3). Many of the decay products of U-238 and U-235, such as lead-214 and bismuth-214, emit gamma radiation and therefore account for a great deal of natural background radiation. The direct decay products of the radioactive noble gas radon (another decay product of U-238) form the main natural source of radiation to which we are exposed, since inhalation of these decay products cause exposure of our lung tissues to alpha particles.

Around the world, a great deal of testing has been undertaken to determine the concentrations of natural radionuclides in the various environmental compartments. Summaries of the latest data are published on a regular basis by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR).[2122,23,24]

3.1 Soil

Published data on uranium in the soil normally give only the activity concentration of U-238. However, on the basis of the data presented in table 2-3, the activity concentration of natural uranium (without decay products) can be calculated simply by multiplying the U-238 figure by 2.05. The weight of uranium present can also be

determined from the U-238 activity concentration, since there is 12.4 Bq of U-238 activity for every milligram of natural uranium.

The concentration of uranium in the soil differs greatly from one part of the world to another. In the United Kingdom, for example, U-238 values varying between two and 330 Bq per kilo of dry earth have been reported (the equivalent of between 0.2 and 26 milligrams of uranium per kilo of dry earth).[24] UNSCEAR gives the average U-238 activity concentration as 33 Bq/kg (2.6 mg/kg).[24]

Variation in the Netherlands is not as marked, but the highest levels are nevertheless twenty times the lowest. The terrestrial radiation map of the Netherlands corresponds closely to the geological map: clay and loess are associated with relatively high levels of activity concentration, and sand and peat with low levels.[25] A great deal of data on the natural radionuclide concentrations in the Netherlands' soil have been collected by Köster *et al.* This team found that U-238 activity levels in Dutch soil could be anywhere between 5 and 100 Bq per kilo of dry earth (0.4 to 8 milligrams of uranium per kilo of dry earth).[26] On the Wadden Islands, at the sites of highly localised black sand deposits, concentrations as high as 300 to 700 Bq (roughly 25 to 60 milligrams) per kilo of dry sand were detected. In the various types of clay, the activity concentration was found to vary between 19 and 53 Bq/kg (1.5 to 4.2 mg/kg). Surface sand strata typically had much lower activity levels: 11 tot 15 Bq/kg (0.9 to 1.2 mg/kg). Van de Plassche *et al* determined that the background concentration of uranium in 'standard soil' was 2.92 mg/kg (36.5 Bq U-238/kg).[27]

The figures recorded in the Netherlands are consistent with information published by the UNSCEAR, which suggests that activity levels are generally in the range of 10 to 50 Bq/kg (0.8 to 4 mg/kg).[22] Concentrations in the USA are generally between 7 and 60 Bq/kg (0.5 to 4.7 mg/kg), but here again, extreme figures are sometimes recorded. In phosphate rocks used as a source of fertiliser, activity levels can be as high as 4800 Bq/kg (390 mg/kg).[28]

3.2 Air

Various authors have published data on atmospheric U-238 concentrations in the USA. Golchert *et al* found that in the Chicago area, the concentration was roughly 0.3 µBq/m³[a] (0.02 ng/m³),[29] while a team led by Fisenne recorded figures of 0.7 µBq/m³ (0.06 ng/m³) in New York City.[30] German researchers have reported U-238 levels of 0.3 to 1.7 µBq/m³ (0.02 to 0.14 ng/m³).[24]

[a] Microbecquerels per cubic metre.

The UNSCEAR has estimated that, given an activity concentration of 25 Bq per kilo (1 milligram of uranium per kilo) in the soil and an airborne dust concentration of roughly 50 µg/m³, the atmospheric activity concentration is likely to be roughly 1.2 µBq/m³ (0.05 ng/m³).[22] On this basis, the average adult would inhale 0.01 Bq (0.4 µg) of uranium per year. The most recent UNSCEAR report indicates that for U-238 and for U-234, the characteristic atmospheric activity concentration is 1 µBq/m³ (0.08 ng/m³).[31] This equates to a radiation dose (effective dose) of 0.021 µSv for U-238 and 0.026 µSv for U-234 respectively per year..

No data is available specifically regarding atmospheric uranium concentrations in the Netherlands. However, the National Institute of Public Health and the Environment (RIVM) does measure overall airborne alpha activity associated with long-lived radionuclides. In 1998, the annual average was 81 µBq/m³ (attributable mainly to polonium-210). On the reasonable assumption that the atmospheric concentration of uranium in the Netherlands is also around 2 µBq/m³ (0.08 ng/m³), it would appear that the contribution of uranium to the natural alpha activity in the air is small.[a]

3.3 Water

In the USA, a great deal of data has been collected on uranium concentrations in drinking water.[32] A total of 35 000 surface water sources and 55 000 groundwater samples have been analysed. The activity concentration in surface waters was found to average 40 Bq/m³ (3 mg/m³), with a spread of 0.4 to 25 000 Bq/m³ (0.03 to 2000 mg/m³). The corresponding figure for groundwater was 100 Bq/m³ (8 mg/m³), with a spread of 0.04 to 24 000 Bq/m³ (0.004 to 1900 mg/m³). The surface and groundwater samples tested included 28 000 samples of mains drinking water. The average concentration in the drinking water samples was 75 Bq/m^3 (6 mg/m³), with a spread of 2.6 to 24 000 Bq/m^3 (0.2 to 1900 mg/m³).

In the Netherlands, insufficient data have been published to put a reliable figure on background uranium concentrations in groundwater, surface waters or sea water.[27] However, it is likely that in wooded areas, the uranium concentration in groundwater varies between less than 0.01 mg/m³ and roughly 1.3 mg/m³ (0.1 and 16 Bq/m³ U-238).

The RIVM has looked closely at radioactivity in bottled and non-bottled mineral and spring water.[33] Although uranium was not directly measured, one may deduce from the data on radium-226 that U-238 concentrations in mineral and spring water of Dutch origin are very low – possibly just a few hundredths of a becquerel (i.e. a few thousandths of a milligram) per cubic metre. During the 1980s, radioactivity levels in

[a] Smetsers RCGM, personal correspondence, RIVM, 2001. Data available on request from the Health Council secretariat.

drinking water supplied by public utility companies in the Netherlands were investigated. Tests were carried out to establish, amongst other things, radium-226 concentrations and overall alpha activity levels in 250 samples.[34] Radium-226 concentrations were found to vary between 2 and 8 Bq/m³, on the basis of which the characteristic U-238 concentration in Dutch drinking water may be estimated at between roughly 0.01 and 0.1 Bq/m³ (0.0008 and 0.008 mg/m³).

Finally, it is also interesting to note that Fisenne calculated that the average New Yorker ingested via water the equivalent of 1.2 mBq (0.09 µg) of U-238 and 1.5 mBq (0.12 µg) of U-234 per day.[3530]

3.4 Food

The 1977 UNSCEAR report included data on U-238 concentrations in food obtained from France, Japan, Russia, the USA and the UK.[21] An average person in these countries was apparently ingesting between 7.4 and 18.5 mBq (0.6 and 1.5 µg) of U-238 per day with his or her food.

From this data, the UNSCEAR estimated the average internal radiation dose arising from the intake of natural uranium. To this end, reference concentrations for U-238 in drinking water and milk products, meat products, vegetables and fruit, grain products, and fish were set at, respectively, 1, 2, 3, 20 and 30 mBq/kg (0.08, 0.16, 0.24, 1.6 and 2.4 µg/kg). By linking these figures to consumption data for the various types of food published by the International Commission on Radiological Protection (ICRP),[36] the average age-weighted annual intake was calculated to be approximately 5 Bq U-238, or 0.4 milligrams of uranium.

Uranium concentrations have not been determined for foodstuffs in the Netherlands in the context of either an ongoing monitoring programme or a one-off project.

3.5 Human tissue

Fisenne's research team published a survey of uranium concentrations in human tissue, based on data from twenty-three studies conducted in twelve different countries (which did not include the Netherlands).[37] It is not possible to determine the distribution of uranium between the various organs for people in any given country; nevertheless, the data does give a general idea of how much uranium finds its way into each of the major organs. Table 3-1 shows the spread of uranium concentrations in the various tissues studied and the average concentrations calculated by Fisenne *et al.* Of the twelve countries for which data is available, it is reasonable to suppose that, given its

geographical proximity, the UK corresponds most closely with the Netherlands. The data for the UK is therefore shown separately in table 3-1.

Table 3-1 Uranium concentrations in human blood and tissue (µg uranium per kg wet tissue,bone ash or blood). The figures in the second and third columns are averages for twelve countries, while those in the last column relate to London.[37]

Organ	Range of U-concentration	Mean U-concentration (± 1 standard deviation)	U-concentration in England (London) (± 1 standard deviation)
Lungs	0.5-6.8	2.5 ± 2.5	
Liver	0.12-11	2.3 ± 4.0	0.25
Kidneys	0.34-5.5	2.3 ± 2.3	
Muscle	0.19-7.7	3.7 ± 4.0	0.19
Fat	0.6-1.9	1.3 ± 0.9	0.6
Blood	0.1-1.3	0.58 ± 0.44	0.69 ± 0.45
Bone	0.10-7.1	2.4 ± 2.3	3.5 ± 0.9
Bone ash	0.4-51	11 ± 11	23 ± 6

The highest concentrations in bone tissue were found in Nigeria; the highest figures for all other tissues and blood were recorded in the former Soviet Union. The ratio between the concentration in bone and that in blood was between 5.1 and 0.2, while the ratio between liver and kidney tissue concentrations varied from 0.5 to 3.3. The average uranium concentration in bone was lower than the 5.9 µg/kg estimated for the ICRP's 'reference man'.[36] From the data in table 3-1, it is clear that the uranium concentration in bone ash varies considerably, the highest figure being 125 times greater than the lowest. This variation cannot be explained simply by differences in the uranium concentrations found in food, where the upper and lower values differ by a factor of three. It is more likely to be attributable to the large spread found in uranium concentrations in drinking water.[5] The authors of the RAND publication also point out that it is known from research in New York City that uranium concentrations in the vertebrae and lungs increase markedly with age.[30] This is not the case, however, where liver and kidney tissues are concerned. Accumulation in the lungs may be due to the barely-soluble inhaled uranium, which remains in the pulmonary lymph nodes for longer periods.

On the basis of the average concentrations for the twelve countries listed in table 3-1, Fisenne calculated the representative annual absorbed dose rate associated with irradiation by alpha particles from uranium for each of the main organs. The results are presented in table 3-2.

Table 3-2 Absorbed doses to organs associated with irradiation by alpha particles from uranium. Dose conversion factors applied (µGy per year per microgram of uranium per kilogram of tissue): for ribs and vertebrae, 0.056; for femur, 0.072 and for soft tissue, 0.54.[37]

Organ	Dose ($\mu Gy.y^{-1}$)
Lungs	1.4
Liver	1.2
Kidneys	1.2
Muscle	2.0
Bone surface	0.62

3.6 Human excreta

The presence of uranium in the human body as a result of inhalation or ingestion leads to its excretion in urine and faeces. Interpretation of data on activity concentrations in the excreta of occupationally exposed workers or other people suspected of inhaling or ingesting uranium from non-natural sources depends on having accurate information regarding the background levels of uranium excretion. These levels are to a large extent determined by uranium concentrations in the local food and drinking water supplies. According to ICRP Publication 23 (which appeared in 1975), the characteristic uranium excretion value for urine is 0.05 to 0.5 µg (0.6 to 6 mBq U-238) per day (0.04 to 0.4 µg/l; 0.5 to 5 mBq/l U-238), while the corresponding figure for faeces is 1.4 to 1.8 µg (17 to 21 mBq U-238) per day.[36] A considerably lower average uranium concentration – 12.8 ng/l (approximately 0.2 mBq/l U-238) – has been found, however, in the urine of people living in an area of India with normal background levels of uranium.[17] In Belgium, Hurtgen has studied uranium excretion by people suspected of having suffered exposure.[18] Between 1991 and 1999, he made alpha-spectrometric analyses of thirty-nine faeces samples and 1143 urine samples to determine their uranium content. In the twenty-four-hour urine samples, the average overall level of uranium activity was 0.3 mBq; the corresponding figure for the faeces samples was 57 mBq. Although Hurtgen's subjects may have included a number of contaminated individuals, the values obtained are consistent with the ICRP's figures.

Spencer *et al* looked at uranium ingestion and excretion in a group of subjects who were monitored for periods of twenty-four to fifty-four days.[38] Little variation was found in the amount of uranium ingested by the subjects with their food, but the amount ingested with drinking water varied considerably. The excretion of uranium in the urine proved to be proportional to the level of ingestion with water. It would therefore appear that water is more significant in relation to the absorption of uranium than food.

3.7 Summary of the situation in the Netherlands

The uranium concentration figures presented in the previous sections of this chapter are derived largely from data collected in other countries. Very little specifically Dutch data is available from which conclusions may be drawn regarding the amounts of uranium normally present in the various environmental compartments or in food. We do nevertheless have a reasonably good picture of the concentrations present in the soil of this country.

3.8 Summary of the global situation

Data on the radionuclide intake associated with fluid ingestion, food ingestion and inhalation has been distilled from the 1993 UNSCEAR report and is presented in table 3-3.[23]

Table 3-3 UNSCEAR reference values for uranium intake by adults, associated with food ingestion, fluid ingestion and inhalation.[23]

	Food consumption (kg/year)	Activity U-238 (mBq/kg)	Concentration nat. uranium (µg/kg)	Intake U-238 (mBq/year)	Intake natural uranium (µg/year)
Milk products	105	1	0.1	105	8
Meat products	50	2	0.2	100	8
Grain products	140	20	1.6	2800	226
Leafy vegetables	60	20	1.6	1200	97
Roots and fruits	170	3	0.2	510	41
Fish products	25	30	2.4	750	60
Water and beverages	500	1	0.1	500	40
	Breathing rate (m^3/year)	**Activity U-238 (µBq/m^3)**	**Concentration nat. uranium (ng/m^3)**	**Inhaled activity U-238 (mBq/year)**	**Inhaled mass natural uranium (µg/year)**
Air	8000	1	0.1	8	0.6
Total intake air plus food				*6 Bq U-238 year*	*0.5 mg natural uranium year.*

As the table shows, the reference value for the intake of U-238 from natural sources is 6 Bq per year (0.5 mg). Given that we are dealing here with natural uranium, and in view of what we know about the activity concentrations of U-234 and U-235 (see table 2-3), it follows that the total amount of uranium activity taken in must be 12 Bq (0.5 mg U). It should be emphasised, however, that the ingestion and inhalation of activity from uranium decay products – in particular radium-226, lead-210 and polonium-210 – will be considerably higher.

Chapter **4**

Exposure to depleted uranium

Exposure to depleted uranium can only result from human activity. The committee feels it is important to distinguish between occupational exposure to DU and exposure experienced by the general public as a consequence of the presence of DU in the environment. The latter is almost always due to the uncontrolled release of DU – in quantities that may vary from a few dozen kilos to several hundred kilos – either in the context of a fire (as in the Bijlmer disaster) or in the context of military activities (as in the Gulf War). Any exposure to DU that an individual experiences is on top of exposure to natural uranium, which is always present in the environment. Where the general public is concerned, the principal mechanisms of exposure to natural uranium are the consumption of food and drink and the inhalation of particles; the levels of exposure involved are summarised in tabl3 3-3. It is important to bear in mind that such exposure entails the intake of natural uranium and all its decay products (see annex B). These decay products are more significant in relation to the committed effective dose[a] than the uranium itself. In this regard, the figures given in table 4-1 for the inhalation of barely soluble uranium compounds serve as a useful illustration.

[a] See glossary.

Table 4-1 Committed effective dose following inhalation of 1 Bq or 1 milligram of uranium (the dose conversion coefficients are relevant for the general public and S-class compounds).

Type of uranium	Dose conversion coefficient	
	µSv/Bq	µSv/mg
Uranium before processing	57.0	710
Processed natural uranium	12.5	225
Depleted uranium	6.8	140

4.1 Military and other forms of exposure to DU

As indicated in section 1.2, wartime exposure to DU is outside the scope of this advisory report. Nevertheless, the committee has addressed the issue of post-combat exposure following the use of uranium-containing ammunition. In this context, four basic forms of exposure may be distinguished:

- exposure resulting from particulate inhalation;
- exposure resulting from ingestion;
- external exposure to radiation resulting from proximity to or contact with DU-containing objects;
- exposure resulting from DU absorption through wounds.

The level of risk associated with exposure depends upon the amount of material involved, whether exposure is acute or chronic, the exposure route, the size of the uranium particles and aerosols involved and, finally, the valency and solubility of the uranium compounds.

4.1.1 Exposure resulting from inhalation of particles and aerosols

When DU shells are used – or, indeed, whenever metallic uranium is burned – only 10 to 35 per cent of the uranium is normally converted to aerosol form. Only in exceptional circumstances will the percentage be higher. Anything between 0.1 and 33 per cent of the particles released during combustion will have a diameter of less than 10 µm. By contrast, nearly all the particles released during the detonation of a uranium shell on impact with a hard object will be less than 10 µm in diameter.[39,40] These particles will be deposited on the ground in the vicinity of the fire or explosion. If disturbed, they can rise into the air again and may thus be inhaled.

4.1.2 *Exposure resulting from ingestion*

DU may be ingested with food or fluids, or as a result of hand-to-mouth contact, especially in contaminated areas. It is also possible to swallow DU-containing aerosol particles floating in the air after an explosion or during a fire. Under such circumstances, ingestion normally continues only for a very brief period, and the uranium is soon excreted from the system. Internal exposure can also result from the swallowing of uranium particles that have settled on the skin or clothing.

4.1.3 *External exposure resulting from proximity to or contact with uranium-containing objects*

As indicated in section 2.3 , in a piece of DU, the concentrations of the relatively short-lived decay products responsible for beta and gamma radiation gradually increase until a state of equilibrium is established a few months after production of the material (see also annex B). External exposure to such radiation is hazardous to human health. The uranium itself emits only alpha radiation, which is not capable of penetrating far enough into the skin to reach any sensitive cells and is not therefore hazardous in the event of external exposure.

Most of the radiation to which the skin and eyes are exposed is attributable to beta particles. At the surface of a DU plate, the dose rate associated with beta radiation is approximately 2.3 mSv/hr, compared with a rate of a few dozen µSv/hr where gamma radiation is concerned, depending on the geometry of the object.

4.1.4 *Exposure resulting from DU penetration through wounds*

Exposure resulting from DU fragments embedded in the body or from the contamination of wounds by DU particles is almost exclusively a military phenomenon. Although it is theoretically possible for DU-contaminated material to come into contact with a wound in a non-military context, normal medical procedures would be sufficient to ensure that only a very small amount of DU entered the body.

4.1.5 *Extent of exposure*

The extent of exposure in a given situation depends on the circumstances under which the DU has been released into the environment and, where occupational exposure is concerned, the activities being undertaken or, where non-occupational exposure is

concerned, the habits of the potentially exposed population. If information regarding these factors is known, it is possible to formulate exposure scenarios and use them to estimate risk levels. This procedure has been followed to assess the risk associated with the use of DU ammunition in Kosovo (see section 4.3 and annex C). Similar analyses have been made in connection with the Bijlmer disaster.[20]

4.1.6 Exposure of children

When considering the risks posed by exposure to DU, it is important to take account of special risk groups such as young children. Children constitute a special risk group for various reasons. For example, the radiation dose received by a child who inhales air with a given concentration of barely soluble uranium compounds will be twice or three times as highas that received by an adult who inhales such air. Furthermore, children playing in a DU-contaminated area are liable to swallow soil that contains DU particles. By way of illustration, an exposure scenario is outlined in annex C.

4.2 Occupational exposure to DU

Exposure to DU can also occur in various occupational contexts, such as the uranium processing industry and other industries in which DU is used. In all such situations, Dutch health and safety legislation and radiological protection regulations require that exposure should be minimised and certainly kept within the applicable limits.

4.3 Exposure in Kosovo

4.3.1 Findings

Following the use of DU-containing ammunition during recent Balkan conflicts, a UNEP delegation conducted tests during a mission in November 2000 at eleven sites in Kosovo; these sites together represented roughly 12 per cent of all locations targeted with DU ammunition.[3,4,14] The potential for contamination associated with the ammunition in question related primarily to barely soluble uranium compounds. On the basis of the tests conducted by the delegation, the UNEP drew the following conclusions regarding the possibility of people in the area being exposed to harmful radiation:

1 No widespread contamination of the soil by DU was detected. In other words, across a larger area, any contamination was too slight to distinguish from the natural background concentration of uranium.

2 In the immediate vicinity (i.e. within a few metres) of DU munition detonation sites, DU contamination was detected.
3 In no case was this contamination sufficient to cause a radiation dose (effective dose) greater than 1 mSv (the limit for the general public). In addition, the radiological risk arising from contact with contaminated sites and the subsequent swallowing of contaminated soil was very slight.
4 No DU-contaminated water, milk, objects or buildings were found.
5 Fallen DU shell heads could in the future contaminate groundwater and drinking water, increasing the overall uranium concentration in the groundwater by between ten and a hundred times. This could in turn lead to levels in excess of the WHO drinking water standard.

4.3.2 Risk assessment

As well as measured data, the 1999 UNEP report included a risk assessment for a reference case.[3] The scenario and assumptions upon which this assessment was based were as follows:

During a raid by three aircraft, a total of ten kilos of DU was fired at one or more vehicles, resulting in the contamination of a thousand square metres of land. The radiation doses associated with this scenario are summarised in annex C. A person spending two hours in the affected area would receive an effective dose of between 0.1 and 10 µSv. It is most unlikely that anyone living in proximity to one of the target vehicles would be permanently exposed to windblown or disturbed dust from the detonation site, but in such an eventuality, the person concerned would receive an annual radiation dose of around 1 mSv.

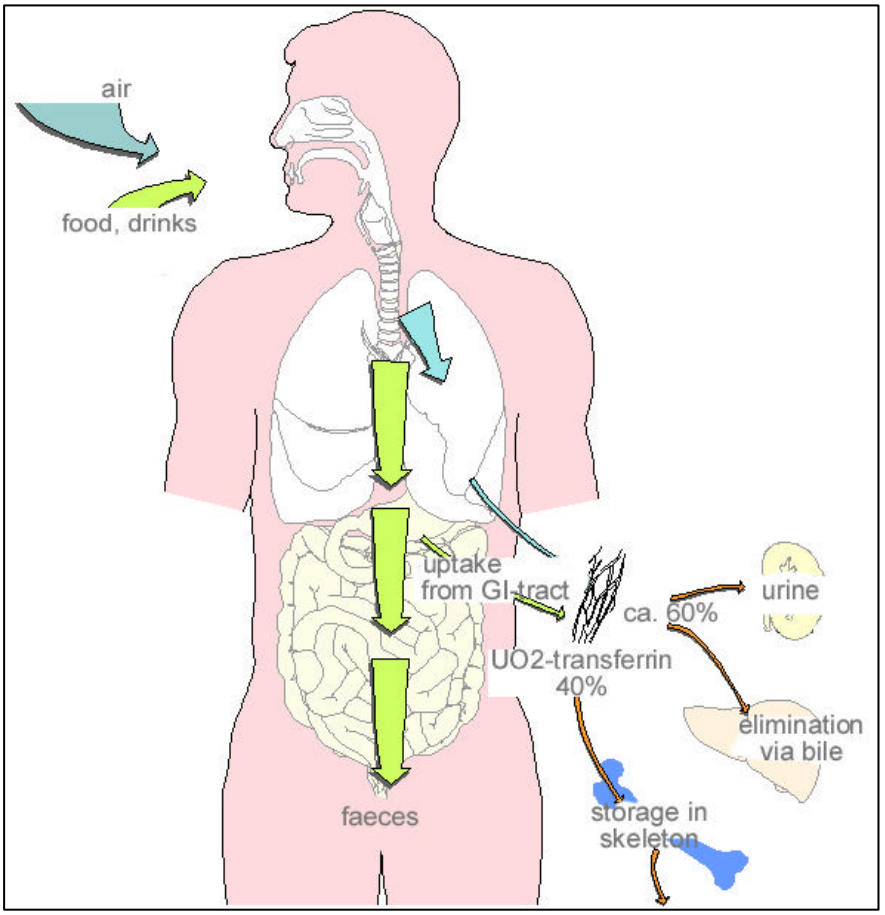
Brenk Systemplanung has developed a similar scenario, which is used for the assessment of DU-related issues by the committee of experts set up under the Euratom treaty.[41]

Chapter 5

Absorption, distribution and excretion

An understanding of the way uranium is absorbed, distributed and excreted by the body is necessary not only for the formulation of safety criteria, such as annual intake limit values for uranium, but also in order to assess exposure following the intake of a quantity of uranium. Models of these processes have accordingly been developed by organisations such as the ICRP.[42,42,43] Such models are built around schematic descriptions of the lungs, the gastro-intestinal tract and the remainder of the body.

In this chapter, the committee describes the processes involved in the absorption, distribution and excretion of uranium. By way of illustration, an explanation is given of the way ingested or inhaled uranium is distributed inside the human body before being excreted. It is important to bear in mind that the fate of uranium following absorption depends to a considerable extent on the chemical form prior to inhalation or ingestion.



Figuur 5-1 Intake, distribution and excretion of uranium by the body.

5.1 Inhalation

Many inhaled aerosol particles remain only briefly in the respiratory system before being exhaled. Some, however, settle in the airways. The point where a particle comes to rest depends on its size, which is normally expressed in terms of aerodynamic equivalent diameter (AED). Some 95 per cent of particles with an aerodynamic equivalent diameter greater than 10 µm that are not immediately exhaled settle in the upper respiratory organs. Most such particles lodge in the pharynx and are subsequently swallowed or expelled through the nose. Smaller particles, however, come to rest in the lungs. Particles of less than 0.5 µm are primarily deposited in the

alveoli, where some are dissolved quickly, others are dissolved more slowly and yet others are almost not dissolved.

Passage from the lungs into the bloodstream depends on the solubility of the compound in question. Uranium oxides and metallic uranium, for example, do not dissolve easily and can consequently remain in the lungs for years. More soluble compounds, on the other hand, can enter the bloodstream in a matter of days or even hours.

To facilitate the estimation of radiation burden following the inhalation of radioactive material, the ICRP has developed a lung model.[43,44] According to this model, given an average particles size of 5 µm, roughly three-quarters of inhaled uranium is immediately exhaled again, while the remaining quarter or so remains in the lungs. The split may be different where aerosols with a different particle size distribution are concerned. Of the particles that are not exhaled, 80 per cent are 'coughed up' and thus enter the gastro-intestinal tract. Nearly all of this material passes through the tract and is excreted; only a small proportion enters the bloodstream. Of the roughly 20 per cent of non-exhaled uranium that does not enter the digestive system, about three quarters is absorbed by the lymph nodes, while the other quarter enters the bloodstream. Thus, less than 1 per cent of all inhaled uranium ultimately finds its way to the kidneys.

The ICRP model distinguishes between three classes of compounds on the basis of solubility: F, M and S (fast, medium and slow), reflecting the rate of elimination from the lungs (see table 2-2). Uranium oxides are in class S, while most hexavalent compounds (e.g. UF_6) are in class F. Residence time in the lungs is a determinant of the radiation dose, not merely to the lungs, but to the entire body (effective dose). Hence, if a person inhales 1 Bq of F-class U-238 compounds, the committed effective dose[a] received will be 0.58 µSv, but if barely soluble S-class compounds are inhaled in the form of aerosol particles with an aerodynamic equivalent diameter of 5 µm with 5.7 µSv/Bq, the dose will be an order of magnitude higher.[b]

5.2 Ingestion

The toxicity of an ingested uranium compound depends largely on its solubility in water, since this determines the ease with which it is absorbed from the gastro-intestinal tract. Uranium oxides are not readily soluble and do not therefore pass easily through the intestinal wall, but pass through the system and are excreted in the faeces. Absorption from the gastro-intestinal tract is also influenced by particle size. Leggett

[a] See glossary.
[b] Data calculated for occupational exposure.

and Harrison have estimated that, on average, roughly 1 to 1.5 per cent of the uranium naturally present in food and fluids ingested by an adult passes through the intestinal wall.[45,46]

In view of the radiological aspects of uranium in the body, the ICRP[42] uses a modified version of the Eve model to predict absorption following ingestion.[47] In this model, passage from the gastro-intestinal tract into the bloodstream is described by a single parameter (f_1), the fraction that is absorbed from the gastro-intestinal tract. This parameter – which is also referred to as the biological availability – depends on the subject's age and dietary habits and on the chemical and physical form of the ingested uranium. According to the model, the amount of uranium that finds its way to organs such as the large intestine, liver and kidneys is proportional to f_1. The values most commonly used for f_1 are:[42]

- 0.02 (2 per cent) for F-class compounds (most readily soluble hexavalent compounds, such as UF_6, UO_2F_2 and $UO_2(NO_3)_2$) and M-class compounds (the less readily soluble compounds, such as UO_3, UF_4, UCl_4 and most hexavalent compounds that are not in class F)
- 0.002 (0.2 per cent) for S-class compounds (barely soluble compounds, such as UO_2 and U_3O_8).

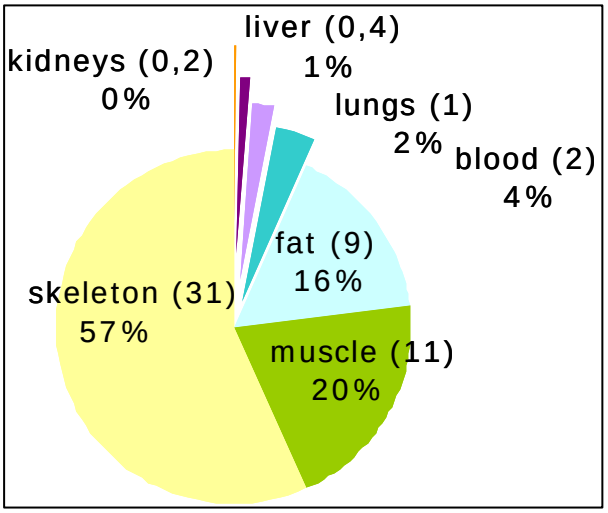
The radiation dose (committed effective dose) of barely soluble S-class compounds per ingested becquerel of U-238 is 0.0076 µSv – an order of magnitude lower than the corresponding figure for readily soluble F-class compounds (0.044 µSv/Bq).[a]

5.3 Distribution

Because natural uranium is continually entering the body – primarily with fluids and food – equilibrium is established between intake and excretion. As a result, the distribution of (natural) uranium concentrations inside the body remains more or less constant. The actual distribution pattern in any given individual depends on the uranium concentrations in his or her food and drink and on certain personal characteristics. The situation is illustrated in Figure 5-2.

Uranium absorbed through the gastro-intestinal tract or the lungs enters the bloodstream and is then either deposited in tissues (primarily bone tissue) or excreted in the urine. It is likely that quadrivalent uranium is oxidised in the body to form hexavalent uranium.[48] Uranyl ions readily form stable complexes with bicarbonate, carbonate and phosphate ions and with proteins, in particular transferrin.[49]

[a] Data calculated for occupational exposure.



Figuur 5-2 Distribution of uranium in the human body. Between brackets relate to mass in µg. [49]

Uranium that is not attached to transferrin is filtered out in the glomeruli of the kidneys, and thus enters the tubular fluid. Positively charged uranium ions can attach themselves to negatively charged receptors (phospholipids) around the striated fringes of the proximal tubular cells. By a process of endocytosis, the uranium is absorbed into these cells and then transported elsewhere in the lysosomes. Some uranium may subsequently be transferred from the lysosomes to other organelles, such as the nucleus and the mitochondria. The amount of uranium that finds its way into the urine depends on the acidity of and the concentration of bicarbonate in the tubular fluid. The less acidic the urine, the more significant the dissociation of the UO_2-bicarbonate complex becomes and the more uranium (in the form of positive ions) is able to attach itself to the membranes of the tubular cells.

The uranium-bicarbonate complex circulating in the blood [$(UO_2HCO_3)^+$] does not dissociate in soft tissues and is not therefore deposited in such tissues. In bone, however, the complex decomposes.[50] Hence:

- uranyl ions can be found anywhere where there is osseous fluid, such as in the canaliculi;[a]
- uranyl ions can participate in ion exchange processes on mineralised bone surfaces;
- following accumulation at growth sites in bones, uranyl ions can become covered by new bone.[46,51]

[a] Small channels through the bone.

Consequently, uranium that is not excreted in the urine is liable to accumulate in the bones.

Uranium is quickly eliminated from the blood: 99.9 per cent is extracted within twenty-four hours. Following intravenous injection of uranyl compounds, half were found to be excreted in the urine and the rest retained in tissues (22 per cent in the skeleton and 21 per cent in the kidneys).[52] Thus, the maximum half-life is five hours.

However, calculations made using the ICRP's kinetic model indicate half-lives for elimination from the kidneys and bone tissue of six days and 1500 days, respectively. This suggests that the rapid elimination from the blood seen after intravenous administration is largely attributable to the diffusion of uranyl ions from the blood, into organs and tissues. Once equilibrium is reached between the concentrations in the bloodstream and in body tissues, the rate of elimination will reduce substantially, since the uranium is distributed over a much larger volume. As a result, chronic exposure can lead to accumulation in the kidneys and in bone tissue. Where uranium is inhaled or taken in orally, the concentration development will be different, since the resorption rate is the major determinant of change in the concentration of uranyl ions in the blood under such circumstances. Consequently, chronic exposure through inhalation or ingestion will lead to a very different pattern of distribution between the various tissues to that associated with acute exposure by means of intravenous injection (Figure 5-2).

Leggett has suggested that the rapid elimination of uranium from the blood seen in the first minutes after injection in the course of experimental research is attributable to the rapidity of filtration in the kidneys and the high speed of diffusion from plasma to extracellular fluid.[53] Thereafter, the rate of elimination slows, due to a combination of factors:

- attachment to plasma-proteins that are capable of binding uranium, are not filtered out by the kidneys and exhibit little extracellular mobility;
- attachment to red blood cell membranes;
- the return of extracellular uranium to the plasma.

Little is known about the relationship between the absorption of uranium from the gastro-intestinal tract and the concentration of uranium in the blood. Leggett theorised that, given the ingestion of 1.75 µg of uranium per day and 2 per cent absorption from the gastro-intestinal tract, the uranium concentration in the blood would be 0.6 ng/l.[53]

A great deal of experimental work has been done with a view to tracking the distribution of uranium following brief exposure based upon administration by intravenous[52], intraperitoneal[54] and oral[55,56] means. Occupational exposure typically involves prolonged inhalation of uranium-contaminated workplace air. Kathren *et al* have looked into the distribution of uranium in the tissues of a fifty-year-old man who

died of heart failure, having previously been exposed to uranium in the course of his work.[57] The man had for many years worked as an operator in the uranium processing industry, and had been exposed to both depleted uranium and 4 per cent enriched uranium. The uranium concentrations in his urine were between 5 and 20 µg/l; excretion via the urine had been reasonably constant over a thirteen-year period and was estimated at an average of 4 milligrams of uranium per year. After his death, uranium concentrations in the man's skeleton, liver and kidneys were in the ratio 63:2.8:1. From in vivo chest count data collected in the ten years prior to death, it was calculated that the total amount of uranium in the man's body was twice the overall amount detected in his lungs and lymph nodes after his death.

Uranium concentration data recorded for bone and other tissues is presented in table 3-1. The concentrations are linked primarily to the ingestion of uranium with drinking water and food. Inhalation hardly contributes to the concentration levels at all. The characteristic amount of uranium in a 'standard person' weighing 70 kilos is 90 µg.[36]

5.4 Excretion

Data was presented in section 3.6 relating to the concentrations of uranium found in urine under normal circumstances. In people who live in areas with normal background concentrations, the average urinary concentration is approximately 15 ng/l (0.2 mBq/l U-238).[17] The ICRP works on the basis of a characteristic concentration of between 40 and 400 ng/l (0.5 to 5 mBq/l).[36]

In the early days of uranium exploitation, scientists began seeking to establish a relationship between the uranium concentration in inhaled air and the uranium concentration in the urine of exposed workers. The research was concerned with factories where exposure was to insoluble UO_2 and soluble UF_4 and UF_6. Lippman *et al* found that, where uranium in soluble forms was concerned, the urinary concentration at the end of the working week was in proportion to the concentration in the air; the concentration ranges were approximately 25 µg/l to 1000 µg/l in urine and 10 µg/m³ to 500 µg/m³ in the air. No such association was detected, however, where workers exposed to barely soluble uranium were concerned.[58]

As indicated in 5, uranium that finds its way into the kidneys is excreted in two phases. In the rapid initial phase, 70 per cent of the uranium is excreted within the first twenty-four hours; a slower phase then follows, during which the metabolic half-life is several months. Under normal day-to-day absorption conditions, the biological half-life of uranium is believed to range from 180 to 360 days.[48] By way of illustration, the RAND report[5] gives the example of a worker who inhaled barely soluble powdered

metallic uranium following an explosion. Over the first ten days following exposure, uranium excretion in the urine declined very quickly, from more than 2000 to approximately 30 µg/l; thereafter, the rate of decline eased markedly, with the concentration falling to roughly 10 µg/l seventy-two days after the explosion.[59]

In the exceptional circumstance of uranium being continuously absorbed by (former) military personnel from DU fragments embedded in the body, the urinary uranium concentration is permanently raised. In 1993/1994, the excretion of uranium by military personnel with DU fragments was 4.47 µg/g creatinine, compared with 0.03 µg/g for a control group. In 1995, the figures were 6.40 and 0.01 µg/g creatinine, respectively.[60]

In summary, when uranium enters the lungs or gastro-intestinal tract or comes into contact with the skin, some of it is absorbed by the body. After entering the bloodstream, a proportion of this uranium is quickly excreted in the urine. The remainder is mainly deposited in the bone.

In the general public, day-to-day absorption and excretion of uranium remains fairly constant. This is not so – or, at least, is less so – where occupational exposure is concerned.

Chapter 6

Health implications of exposure to depleted uranium

The health implications of exposure to natural or depleted uranium derive not only from the substance's radiotoxicity, but also from its chemical toxicity. Exposure to radiation is associated most closely with the possibility of an increased risk of cancer – particularly lung cancer following the inhalation of uranium-containing compounds and bone cancer following the ingestion of such substances. As with other heavy metals, it is the kidneys and liver that are most likely to be affected by uranium's chemically toxic properties. Uranium was first suspected of causing glucosuria [a] and other kidney dysfunctions as early as about 1850.[61]

A great deal of epidemiological research has been conducted into the effects of occupational exposure to natural (and enriched) uranium. This research has been reviewed in various surveys.[5,6,7] Exposure to DU can be expected to affect the health in the same way as exposure to natural uranium. Most of the studies have focused on people who were exposed to increased uranium concentrations over very long periods as a result of working in the uranium processing industry. In addition, a number of cases of acute exposure to uranium caused by industrial accidents have been reported. Numerous animal studies have also been carried out to determine the effects of uranium. A thorough survey of such studies was published by Hursh and Spoor in 1973.[62]

[a] Raised glucose levels in the urine.

6.1 Cancer

Uranium is supposed to have both chemical and radiotoxic properties that may cause cancer. The reactive uranyl ion can trigger the formation of compounds that are capable of damaging the DNA. The results of research in this field are discussed in the RAND report.[5] The report's authors conclude that, while most studies have failed to find evidence of structural damage on a histological level, *in vitro* research[a] indicates that some uranium compounds – uranyl nitrate, for example – are genotoxic.

It is known that ionising radiation can induce cancer. Research has found increased rates of chromosomal abnormality and DNA damage – phenomena that are consistent with increased cancer risk – amongst uranium mine workers. However, it is likely that these effects are attributable to radioactivity from sources other than the uranium itself, in particular radon decay products. Other factors, such as smoking and potentially genotoxic micro-organisms (*Aspergillus* and *Penicillium*) may also play a role.[7]

The authors of the *Toxicological Profile for Uranium* conclude that there is no evidence to support the assumption that exposure to depleted or natural uranium through ingestion is a cause of cancer in humans.[7]

6.1.1 Lung cancer

Dogs and monkeys exposed to high atmospheric uranium concentrations sometimes developed slight fibrosis in the tracheobronchial lymph nodes; similar changes were also seen in the lungs of the monkeys. The radiation doses (absorbed dose) from alpha particles were in these cases more than 5 Gy in the lungs and 70 Gy in the lymph nodes. Lung fibrosis is to be expected after brief high-dose rate exposure.[5] A small but significant increase in the incidence of lung cancer was observed in dogs after prolonged exposure to natural uranium oxide by inhalation.[63]

Most epidemiological studies have failed to establish any link between exposure to uranium and lung cancer mortality rates. Many of these studies followed large numbers of workers over considerable periods. Dupree *et al* conducted a patient-control study, taking in 787 cases of lung cancer in workers at four companies where uranium was handled.[64] The findings suggested that a lung dose (absorbed dose) of up to 250 mGy did not increase lung cancer risk. Exposure to higher doses did, however, appear to be associated with an increased risk of lung cancer, but the number of cases was too small to support any definite conclusions. Ritz reported a statistically significant rise in

[a] Research on cells and tissues.

mortality due to lung cancer in a small group of people who were estimated to have received cumulative lung doses of roughly 200 mGy[a] or more.[65] No association was found at lower doses. It should be noted, however, that Ritz's findings were based on just three additional cases of lung cancer, and that Ritz was not able to make any correction for smoking habits.[6] People who inhaled uranium in an insoluble form during military operations or following uranium fires were not found to be more likely to contract lung cancer. The only uranium-related increase in lung cancer risk that has been reported involves people exposed to enriched uranium.[5]

A committee of the American Institute of Medicine recently concluded – partly on the basis of the research referred to above – that the epidemiological evidence tended to suggest that the inhalation of uranium was *not* associated with an increased risk of lung cancer, at least where the lung dose was less than the 200 mGy level mentioned above. Where higher cumulative levels of exposure were concerned, the epidemiological data was inconclusive regarding any association between exposure to uranium and the incidence of lung cancer.[6]

6.1.2 *Skin cancer*

There is no epidemiological evidence that exposure to uranium can cause skin cancer. The increased incidence of skin cancer in Czech and Chinese uranium mine workers is probably due to exposure to arsenic.[6]

6.1.3 *Bone cancer*

As indicated earlier, bodily accumulation of uranium occurs in the bones. It has been calculated that the typical daily intake (see table 3-3) of 1.4 µg (35 mBq) with food and drinking water results in an annual alpha dose to the skeleton of 0.25 µGy.[37] Assuming that there is no threshold dose and that a linear relationship exists between radiation dose and cancer risk, lifelong exposure to such an annual dose would cause two additional cases of bone cancer (osteosarcoma) per million individuals. An increase of this order would not be demonstrable in practice (in the Netherlands, the incidence of bone cancer is approximately four per million residents per year). Moreover there are in fact indications that this particular form of cancer is not induced by alpha particle doses of less than about 10 Gy.[5]

[a] In the paper by Ritz *et al*, the lung doses are expressed in millisieverts. Upon enquiry, however, the committee established that the authors had actually converted absorbed dose data in expressed grays to sievert equivalents, using a radiation effectiveness factor of 1. The radiation effectiveness factor of alpha particles is, however, considerably greater than 1.

6.1.4 *Leukaemia*

Several studies have been conducted into the induction of leukaemia in people working in the uranium processing industry.[7] Only in one such patient-control study was any increase in leukaemia-related mortality noted.[66] However, the subjects in question had also been exposed to external gamma radiation with a cumulative dose in the range of 1.2 to 193 mSv, making it difficult to distinguish the effects of uranium. The situation was further complicated by the fact that some of the subjects had additionally suffered internal contamination not only by natural uranium, but also by fission products, plutonium and enriched uranium. Moreover, a discernible increase in the incidence of leukaemia following inhalation of insoluble uranium compounds is not particularly plausible in view of the low bone marrow dose that would result – less that one thousandth of the committed dose to the lungs (0.049 and 67 µSv/Bq U-238 respectively[a]). The likelihood of leukaemia induction per inhaled quantity of insoluble uranium would therefore be more than a thousand times less than the likelihood of lung cancer induction. The committed dose of soluble compounds is even smaller than the dose of barely soluble compounds.

6.1.5 *Other forms of cancer*

In addition to the forms of cancer considered above, researchers have looked into the possibility of a link between exposure to depleted uranium and the incidence of other malignant conditions. The RAND report indicated that rats that had been exposed to highly enriched uranium (92.8 per cent U-235) were susceptible to various forms of cancer, including leukaemia, lung cancer and renal cancer.[5] Since DU is much less radioactive than enriched uranium, these findings shed little light on the implications of exposure to DU. The development of cancer of the lymph nodes has been linked to the inhalation of uranium.[7] A study of 2002 uranium industry workers found six cases of death from this form of cancer, where on average only 2.6 cases would have been expected.[67b] The latency period was twenty years. In another study of uranium workers, a slight increase in the incidence of death from tumours of the lymphatic and haematopoietic tissues was observed.[68] The researchers attributed this to irradiation of the lymph nodes by thorium-230, a decay product of U-234 and thus part of the U-238 decay series, which is all but absent in DU (see also annex B).

[a] According to the ICRP, the lungs and the bone marrow are equally sensitive. See also annex D.

[b] As far as the committee is aware, this data has not been published in peer reviewed literature.

6.2 Kidney disease

In numerous studies where dogs, rats or rabbits were exposed to uranyl salts (amongst other things), acute kidney dysfunction was observed. Sensitivity to these compounds varies considerably, rats apparently being much more sensitive than rabbits, for example. Gartland *et al* observed dose-related effects following the administration of uranium to rats in concentrations of 3, 6 and 12 milligrams of uranium (in the form of uranyl nitrate) per kilo.[69] As the dosages increased, the concentrations of citrate, creatinine and 2-oxoglutarate in the urine fell and concentration of 3-D-hydroxybutyrate rose.

Researchers have also observed mortality in laboratory animals due to kidney failure following the inhalation of uranium compounds, albeit at concentrations that were very high by practical standards. After exposure to a uranium concentration of 8.1 g/m³ for ten minutes, 50 per cent of the rats in a study died from renal failure.[7,70]

Industrial accidents leading to exposure sufficient to cause urinary uranium concentrations to reach levels of around 2 mg/l are known to have caused temporary kidney dysfunction in affected workers.[49] It also appears that the presence of uranium in drinking water may be linked to increased levels of the protein albumin in the urine, although not accompanied by any symptoms of illness.[71] Kathren, who studied kidney tissue obtained from autopsies conducted on uranium industry workers, did not find any signs of renal damage following prolonged exposure to uranium.[57,72]

In 1998, Zamora *et al* published the findings of a study that looked at kidney function after lifelong exposure to natural uranium in drinking water.[73] The group that had received the highest exposure – the study group – had been taking in between 3 and 570 µg of uranium per day, while the control group took in 0.3 to 20 µg per day. The urinary concentrations of various biomarkers[a] did appear to be linked to uranium intake, but were nevertheless within the normal ranges. No correlation was detected between the intake of uranium and protein concentrations in the urine. It was not clear whether or to what extent the data was indicative of permanent damage resulting from uranium ingestion.

The latter observation might be made regarding most of the cross-sectional epidemiological studies published over the last twenty years that have looked at the effects of exposure to potentially nephrotoxic[b] substances. In many cases, a correlation has been detected between exposure and urine composition abnormalities – typically raised concentrations of low-molecular-weight proteins which, following filtration, are

[a] Biomarkers are compounds whose concentration in the body or in excreted products provides information regarding the individual's exposure to a particular substance, or regarding a biological effect caused by a particular substance.

[b] Poisonous to the kidneys.

normally resorbed from the proximal tubules. Some studies have made use of urinary biomarkers – mainly membrane proteins[a]. Only in a few cases – such as a study of people exposed to cadmium – has an attempt been made to determine the extent to which the findings are indicative of possible renal damage (by following up the exposed population over a period of years). The study in question came to the conclusion that raised protein concentrations were not indicative of renal damage.[74] The value of micro-albuminuria as a predictor of diabetic nephropathy is an exception in this regard.[74]

To sum up, animal studies indicate that exposure to uranium resulting from high levels of intake (several milligrams of uranium per kilo bodyweight) affects renal function, while extremely high intake levels lead to acute renal insufficiency. However, when exposure ceases, renal function recovers in most cases. There is no evidence that chronic renal insufficiency is more common among people who undergo chronic exposure to raised concentrations of uranium. Nor are such problems more prevalent among war veterans with uranium-containing metal fragments embedded in their bodies.[60]

6.3 Liver disease

Animal studies have revealed that high uranium doses can damage the liver.[7] One such study was conducted by Domingo *et al.*[75] Sprague-Dawley rats were given single doses of 5.6 and 118 milligrams of uranium per kilo in the form of uranyl acetate dihydrate, following which the animals developed micro-haemorrhage points in the liver.

No reliable data is available on the effects of uranium on the human liver. However, a case has been reported, in which a man deliberately drank 15 grams of uranyl acetate in combination with an unknown amount of benzodiazepine in a suicide attempt.[76] Uranyl acetate is readily soluble in water and is therefore absorbed from the gastro-intestinal tract more quickly than barely soluble compounds. The man in question suffered temporary liver dysfunction, but all signs of liver damage were gone six months after ingestion.

To date, there have been no reports indicating that liver disease might be unusually prevalent in Gulf War veterans or people exposed to uranium in the course of their work.[5]

[a] Proteins from the membranes of cells in a particular segment of the nephron.

6.4 Effects on reproduction

The potential effects of uranium on reproduction and development may be divided into several categories: damage to the reproductive system, changes to the genetic material that can cause congenital abnormalities, and adverse effects on the development of the foetus or baby. Again, it is important to consider both radiotoxicity and chemical toxicity.

The radiation doses to the reproductive organs associated with the intake of uranium are modest by comparison with the size and variability of the natural background dose.[5] This is also true in relation to groups of people exposed to DU, such as military personnel who served in the Gulf War. There have been no congenital abnormalities in the children of (former) military personnel with DU fragments embedded in their bodies; however, since only thirty-three veterans and seventeen births are concerned, this fact is of little statistical significance.[5]

The conclusion is that neither the epidemiological research nor the animal experiments conducted to date indicate that exposure at the levels associated with military or other uses of DU has any effect on reproduction or development or any genotoxic consequences.

6.5 Other effects on health

Little research has been conducted with a view to determining how exposure to uranium might affect the health in ways other than those referred to above. A certain amount of work has nevertheless been done in this field. Changes have been observed, for example, in the blood of uranium miners, such as altered haemoglobin concentrations and red blood cell counts. Other researchers could not confirm these observations, however; the reported values were within the normal ranges and any changes that may have occurred could not be attributed with confidence to exposure to uranium.[5,77]

Researchers focusing on the human immune system and nervous system have failed to detect any effect.[66,78]

One case was reported, which involved a volunteer who ingested a dose of uranyl nitrate of 14.3 mg/kg.[79] Within a few hours, the individual in question was affected by nausea, vomiting and diarrhoea. However, all discernible effects had gone after twenty-four hours.

The authors of the American Institute of Medicine's report concluded that there was insufficient evidence to determine whether there was any link between exposure to uranium and the relevant affections of the nervous system,and the lungs, gastro-

intestinal complaints, immune system dysfunctions, changes in blood composition, cardiovascular diseases, eye problems or muscular diseases.[6]

Acute mortality

Mortality has been observed in laboratory animals following oral administration of high uranium doses.[7] For example, when rats were fed a diet that contained 2 per cent uranium (in the form of UO_3) by weight in a thirty-day study, all of them died.[80] Oral LD_{50} values[a] of 114 and 136 mg U/kg were determined by Domingo *et al* for male rats and male mice following the administration of single doses of uranyl acetate dihydrate.[75]

So far as the committee could ascertain, human exposure to uranium by oral means has never led to death, although cases have been reported where people have inhaled high concentrations of uranium hexafluoride, with fatal consequences. However, the cause of death in these cases was damage to the respiratory organs by hydrogen fluoride, rather than exposure to uranium.[31]

6.6 Contamination of wounds and embedded DU fragments

During the Gulf War, a number of people in the armed forces were hit by fragments of uranium, which have since remained embedded in their bodies. This has led to a demonstrable increase in the excretion of uranium via the urine. Although the individuals in question suffer from a variety of health problems that seem to be directly related to their war wounds, none of them have yet exhibited kidney dysfunction that could be related to the (chemical) toxicity of DU. Nor do any of them display symptoms that might reasonably be attributed to exposure to radiation. A link has, however, been observed between urinary uranium levels and the results of performance tests (scores decreased with increasing uranium exposure).[81]

From a radiological point of view, the fact that DU fragments embedded in the skin can irradiate the basal skin cells is significant, since it is these cells that are most sensitive in terms of the induction of cancer. A fragment of 100 µm, containing 0.15 Bq of uranium activity, is sufficient to kill all cells within range of the alpha particles (approximately 35 µm). Most of the tissue damage caused by embedded fragments or splinters is attributable to alpha radiation. This is in contrast to the situation with uranium that remains outside the body, where the danger comes from beta and gamma radiation only.

[a] LD_{50} – the dose (administered quantity per unit body weight) at which half the laboratory animals die.

From the information presented in this chapter, it is apparent that uranium and uranium compounds can be both radiotoxic and chemically toxic. However, there is no evidence to suggest that uranium is hazardous at the levels of exposure to which members of the general public are normally subject as a result of the ingestion and inhalation of material naturally present in the environment. Nor is there reason to believe that exposure at the levels typical for the uranium industry and people present in war zones can be dangerous. The health problems observed among uranium miners are almost certainly attributable, not to exposure to uranium, but to the inhalation of radon decay products (combined with other adverse environmental factors).

Chapter 7

Exposure limits

Before concluding this report, it is helpful to briefly consider the basis of the existing exposure limits for uranium. These limits have been formulated with the aim of providing protection against the chemical and radiological health hazards associated with exposure to natural and depleted uranium. At the outset, it should be emphasised that, in this context, the term 'limit value' means a level of exposure, above which countermeasures are called for; a limit value is not the boundary between levels of exposure that are and are not harmful to human health. The type of limit value considered here – the individual dose limit – is the highest level of exposure experienced by any individual in a group that is consistent with the objective of minimising exposure within that group as far as is reasonably possible. Where occupational exposure is concerned, a threshold limit value or the maximum acceptable concentration (MAC) of a substance corresponds to the level of exposure below which there will be no adverse effect on the health of an exposed individual (or, where Dutch MACs are concerned, the health of an exposed individual's offspring), even if exposure continues throughout the individual's working life.

7.1 Radiation dose limit

As will be clear from chapter 6, there is insufficient epidemiological or animal research data on the radiological effects of uranium to support health-based recommended

exposure limits. However, the committee endorses the recommendations made in previous Health Council reports, which were based on the principle that the effects of ionising radiation, in particular the induction of cancer, can be assessed on the basis of the radiation dose.[82] This approach implies that any radiation dose carries a certain risk, which for protection purposes can be quantified as an increase in the chance of developing cancer at a later date, following exposure to an effective dose of 1 millisievert, of 5 in 100 000.[83] This figure, which has previously been endorsed by the Health Council,[82] has been calculated on the basis of pessimistic assumptions – i.e. assumptions that are liable to lead to the radiation risk being overestimated, rather than underestimated. In the Netherlands, the total number of cancer deaths per 100 000 members of the population averages between 20 000 and 30 000.

National and international law applies what is known as the ALARA principle: the principle (supported by the Health Council) that exposure to radiation should be as low as reasonably achievable. Individual effective dose limits are set on this basis.[11,82,83,84] The individual dose limit for the general public is 1 mSv a year, while the corresponding figure for people exposed in the course of their work is 20 mSv a year. These limits relate to exposure to all sources of ionising radiation collectively, insofar as such exposure may be associated with human activity.

Interestingly, draft new Dutch regulations specify so-called secondary levels: 1 µSv for the effective annual dose attributable to emissions to water and air, and 10 µSv for the dose attributable to external radiation. Below these levels, application of the ALARA principle ceases to be a priority.[11] Moreover, the equivalent organ doses received by exposed workers in a given calendar year are also subject to limits: 500 mSv for the skin[a] and 150 mSv for the lens of the eye. For the general public, the corresponding limits are ten times lower: 50 mSv and 15 mSv, respectively.

The dose limits referred to above apply, not only in the Netherlands and other EU countries, but also in many other parts of the world. Exposure to depleted uranium is always linked to human activity. As such, it must not lead to contravention of the specified dose limits.

7.2 Limit values based on the chemical toxicity of uranium

Atmospheric concentration

The threshold limit value currently recommended by the American Conference of Governmental Industrial Hygienists (ACGIH) for the concentration of depleted and

[a] Average across 1 cm² of skin.

natural uranium compounds in the air of a workplace is 200 µg/m³ (maximum advisable eight-hour average).[85] This limit, which covers both soluble and insoluble compounds, was put forward in 1976 and is still applied in the Netherlands,[86] but is now being reconsidered in this country. The State Secretary for Social Affairs and Employment has asked the Health Council to review many 'older' MACs, including the MAC for uranium.

The origin of the ACGIH figure is not entirely clear, but it is very probably based on the results of extensive animal research carried out around 1949 at the University of Rochester.[5] The outcome of these studies was that uranium was classified as a toxic substance and a cause of kidney damage. The limit value for atmospheric concentrations is based on two basic principles:[87]

- The amount of uranium in the lungs, kidneys or bone following exposure to atmospheric concentrations below the specified maximum should not represent a radiological risk.
- Characteristic uranium-induced damage to the kidneys is a reliable indicator of chemical toxicity; an atmospheric concentration of a soluble uranium compound that does not result in kidney damage may therefore be regarded as a safe concentration.

Concentration in the kidneys

A commonly applied biological limit value for the concentration of uranium in the kidneys is 3 µg per gram of kidney tissue. This figure is based upon research carried out at the universities of Rochester and Chicago, which did not point to the existence of a clear toxicity threshold for uranium in the kidneys of animals, but did indicate that 2 to 3 µg of uranium per gram of kidney tissue could be accumulated without serious adverse consequences.[61] There was also evidence to suggest that, in humans, concentrations of 2 to 6 µg/g in the kidneys had no serious impact on health. Hence, in 1959, the International Commission on Radiological Protection (ICRP) proposed that, given what was known about uranium's chemical toxicity, the maximum permissible concentration should be 3 µg per gram of kidney tissue.[61] This limit has since been generally adopted, but is more the product of consensus among experts than precise scientific determination.

More than ten years ago, an evaluation was made of the data available on uranium-induced kidney damage, from which it appeared that minor forms of damage were observed in laboratory animals at uranium levels of 0.1 to 0.4 µg/g.[61] The committee accordingly supports Leggett's view[61] that, from a preventive perspective, it would be advisable to reduce the kidney damage guide value to around a tenth of its

present level, at least until more is known about the medical significance of subtle physiological changes induced by uranium.

7.3 Comparison of radiological and chemical limit values

By assuming that a worker inhales 1.2 m³ of air per hour and works two thousand hours a year, it is possible to compare the current radiological and chemical limit values for *occupational* exposure. If the inhaled air contains DU in a concentration equal to the TLV of 200 µg/m³ and the AMAD (Activity Median Aerodynamic Diameter) of the particles is 1 µm, the radiation dose (committed effective dose) will be 53 mSv in the case of barely soluble uranium compounds and 3.6 mSv in the case of soluble compounds. If the AMAD of the particles is 5µm, the corresponding committed dose figures will be 41 and 4.3 mSv, respectively.[11] Hence, as things stand, occupational exposure to barely soluble DU compounds is limited by the radiological protection requirements, since 20 mSv a year equates to an atmospheric concentration of 75 µg/m³ (if the aerosol particle diameter is 1 µm) or 96 µg/m³ (5 µm); the corresponding figures for natural uranium compounds are 44 and 56 µg/m³ or 1.1 and 1.4 Bq/m³, respectively. By contrast, where readily soluble DU compounds are concerned, the chemical limit value of 200 µg/m^3 is decisive.

If, however, a much stricter chemical toxicity-based standard is applied for the atmospheric uranium concentration than the ACGIH's TLV value of 200 µg/m³, or if one assumes that the non-natural radiation dose is not attributable to uranium alone, a very different picture emerges. Nevertheless, this comparison illustrates the committee's essential point, namely that both chemical toxicity and radiotoxicity may be relevant when assessing the significance of exposure to (depleted) uranium. It should be noted in this context that the exposure scenarios considered in this document (chapter 4) all involve exposure to barely soluble compounds, where radiotoxicity is the principal concern.

Chapter **8**

Conclusions and recommendations

As indicated in section 1.2, the committee's remit was to undertake a survey of:

- the health risks of exposure to depleted uranium and
- appropriate precautions for people working or otherwise present in areas where depleted uranium has been released into the environment.

This chapter starts with a summary of the committee's findings regarding the health risks associated with exposure to depleted uranium. Thereafter, appropriate precautions are considered. Earlier in this report, it was pointed out that the consequences of exposure to DU depend largely on the form of the material in question. Under most of the circumstances considered in this report, certainly those involving the exposure of members of the general public, the forms of DU concerned are barely soluble substances, mainly oxides.

8.1 Health risks

8.1.1 Data

A significant body of data pertinent in relation to the health risks associated with exposure to depleted uranium is reviewed in this report (see in particular in chapters 4, 5 and 6). These risks derive from both the chemical- and radiotoxicity properties of

DU. Although comparatively little research has looked specifically at exposure to DU, a great deal of work has been done on the effects of natural and enriched uranium. Such work is relevant in relation to DU, since DU disperses through the body in the same way as natural (or enriched) uranium, and isotopic composition has no bearing upon chemical toxicity. Natural and enriched uranium are more radioactive than DU, so the radiological risk associated with exposure to a given amount of DU is less than that associated with exposure to a similar amount of another form of uranium.

At first sight, studies of people working in the uranium processing industry would appear to be the most promising source of conclusive evidence regarding the health effects of constant exposure to the various forms of uranium. Such research does, however, have certain shortcomings in relation to the assessment of DU. First, although tens of thousands of subjects have been followed up, the populations studied are not large enough to allow small increases to be detected in less common forms of cancer. Furthermore, detailed information regarding the exposure of individual subjects was generally lacking, making it hard to distinguish the effects of exposure to uranium from the effects of exposure to other radionuclides, such as radium and thorium, or to other toxic substances. Reliable data on subjects' smoking habits were also unavailable in most cases, confusing the picture where the relationship between uranium and lung cancer was concerned. Nevertheless, the committee regards research of this type as a valuable source of information.[6]

8.1.2 Radiological effects

The most significant points to come out of epidemiological research into the consequences of human exposure to uranium are as follows:

- (Former) military personnel hit by DU fragments during the Gulf War exhibit raised urinary uranium concentrations. However, it has not been possible to link the health problems suffered by such people to their exposure to radiation.
- No cases of lung cancer have been linked with radiation doses to the lungs of less than 200 mGy. No increases in the incidence of other forms of cancer can confidently be ascribed to the radiological consequences of inhaling or ingesting natural or depleted uranium.
- There is no clear evidence of a uranium-induced increase in the incidence of leukaemia in uranium workers. The absence of any link is consistent with findings obtained by modelling radiation dose distribution within the body; from such modelling, it is apparent that the radiation dose to the bone marrow following uranium compound inhalation is a tiny fraction of the dose to the lungs.

- External irradiation by uranium-containing objects is of concern mainly in relation to the skin and the eyes. The doses to these parts of the body associated with incidental exposure would not exceed the existing limits, however, and should not therefore have any adverse effect on health. Problems are likely only in the event of extremely prolonged exposure of the skin through direct contact with unshielded uranium.

Despite the absence of any epidemiological evidence that exposure to uranium in the workplace or in the general environment is harmful to the health, there is no reason to doubt the generally accepted belief that any exposure to radiation entails an element of risk, in particular a risk of cancer induction.[82,83] At the levels of exposure to uranium typically seen in practice (see chapter 3), the risk is simply too small to be readily apparent from epidemiological data. This is only to be expected, given the relatively low activity concentrations of both natural and depleted uranium. Such low activity concentrations mean that, in practice, it is in practice almost impossible to take in enough uranium to receive a substantial radiation dose. Nevertheless, the committee recommends that, wherever exposure to DU is possible, appropriate precautions should be taken to minimise the radiological risk (see section 8.2).

8.1.3 Toxicological effects

The risks associated with exposure to soluble natural or depleted uranium compounds derive primarily from such substances' chemical toxicity, rather than their radiotoxicity. As exposure increases, the kidneys are likely to be the first organs in which abnormalities develop that are significant in relation to health. By contrast, as indicated earlier, the chief risk from the inhalation of insoluble compounds is radiological.

The committee draws the following conclusions:

- Brief periods of exposure involving several milligrams of uranium can induce changes in the kidneys which, at very high doses, lead to acute but generally reversible kidney dysfunction.
- No dose-related change in the incidence of kidney disease has been observed in population groups experiencing chronic exposure to natural uranium.
- To date, no uranium-induced kidney dysfunction has been detected in people working in the uranium industry or in (former) military personnel, including individuals with DU fragments embedded in their bodies.

The scientific data presently available tend to suggest that exposure to uranium or DU, either in the workplace or in the general environment, does not cause kidney damage, at least not at the concentrations that are found in practice.

8.2 Precautions

8.2.1 Approach

In common with uranium of a natural isotopic composition and with various other heavy metals, DU is a toxic substance and should therefore be treated with care. However, as the previous section makes clear, the risks associated with exposure to DU are not particularly great. In considering appropriate precautions, the committee has therefore confined itself to exposure to DU in circumstances where the material is or has been released into the environment by fire or accident, or by the use of DU ammunition. Distinction is made between exposure affecting the general public and occupational exposure involving aid workers, people engaged in the maintenance of public order or military personnel on peacetime duties. In the Netherlands, other circumstances where occupational exposure to DU occurs or might occur are covered by institutionalised occupational safety and radiological protection regimes. The consideration of such circumstances lies outside the committee's remit. The committee does not believe that exposure of the general public in circumstances other than those referred to above (fires, military operations) is a realistic possibility.

The committee's recommendations assume the existence of an area where soil, water and air may contain concentrations of DU, or where DU debris may be scattered around. Furthermore, the precautions proposed are intended to supplement normal radiological protection measures. Hence, the overall package of precautions should be sufficient to take account both of DU's radiotoxicity and its chemical toxicity.

The standard radiological protection advice may be summarised as follows:

- Avoid unnecessary exposure and reduce unavoidable exposure as far as reasonably possible.
- Ensure that no individual is exposed to an effective dose – from all sources of ionising radiation associated with human activity, not merely from uranium – of more than 20 mSv per year in the course of his or her work or of more than 1 mSv per year under other circumstances.

8.2.2 *Is the environment contaminated?*

Before implementing a package of measures, it is necessary to establish whether contamination with DU has taken place, or whether there is DU debris in the area. If so, appropriate action needs to be taken. First, it is necessary to carefully survey the locations where contamination or debris dispersal has taken place and to measure the DU concentrations at such locations, preferably under the supervision of a radiological expert. The following steps should be considered:

- Mark out the suspect area in accordance with expert advice.
- Determine a level of contamination, below which no restriction of activities need to be imposed.
- Localise the DU fragments and/or DU-containing materials.
- Make arrangements for sampling soil, water, air and food and for comparison of the uranium concentrations with the natural uranium concentrations normal in the area.

8.2.3 *Occupational exposure*

Military personnel and aid workers working in areas where DU has previously been used in combat do not need to be regarded as 'exposed workers' in radiological protection terms, unless, for example, the levels of contamination are unusually high.[11,82,84] Where exceptional circumstances do warrant classifying such people as exposed workers, the levels of external exposure and internal contamination should be determined and recorded. In addition, the individuals concerned should be informed about the risks associated with exposure and about the precautions to be taken.

The committee assumes that under such circumstances, normal radiological precautions will be taken if it is actually necessary to work in contaminated areas and/or with contaminated objects.

Suitable arrangements should be made for checking DU-exposed workers for internal contamination. In the committee's view, such arrangements should include provision for the periodic analysis of twenty-four-hour urine samples. Blood tests are not considered particularly useful, since uranium is quickly eliminated from the blood (see chapter 5) and the analysis of urine is in any case more straightforward than the analysis of blood. Urine samples should be checked not only for the presence of uranium, but also for biomarkers such as albumin, acetyl-glucose aminidase and alkaline phosphatase, which have proven their epidemiological value. The analysis of faeces or checks with total body-counters are not normally necessary. A total body-

counter is appropriate only in exceptional circumstances, where there is reason to suspect a high level of internal contamination.

In the event of a stretch of open land becoming contaminated, as may be the case following the military use of DU, there is a danger of small aerosol particles being disturbed and rising into the air. The RAND report suggests a method for assessing this 'resuspension risk'.[5] Climatic factors, such as wind and precipitation play a major role in the resuspension process, as does the depth of the particulate layer that is in danger of disturbance.

8.2.4 Exposure of the general public

It is not possible to make general statements regarding the necessity of action to limit exposure to DU or regarding appropriate measures to take. What is appropriate will always depend on the individual circumstances: the nature and extent of any contamination of the area in question. However, it follows from the information presented in the body of this report that drastic measures will very rarely be required.

The possible presence of DU can engender public disquiet and anxiety, as was seen in the Netherlands following the Bijlmer disaster.[19,82,84] For this reason, the committee considers it vital that, as soon as it appears that DU may be present in an occupied area, local people are informed about the risks and precautions that are being and will be taken. In this context, the committee refers readers to a recent Health Council report, which suggested action to be taken where concerns exist regarding the health implications of environmental pollution.[88] The procedures referred to in that report are equally appropriate in cases where DU contamination is suspected.

The Hague, 16 May 2001,
for the committee

(signed)

prof. dr WF Passchier,	ir JWN Tuyn,
voorzitter	secretaris

Literature

1 Nato Confirms To The United Nations Use Of Depleted Uranium During The Kosovo Conflict. Geneva: United Nations Environmental Programme, 2000; UNEP News Release 00/33. Internet: http://www.unep.org/Documents/Default.asp?DocumentID=80&ArticleID=1549.

2 Beusekamp W. NAVO-munitie wellicht oorzaak kanker. De Volkskrant 23-12-2000.

3 UNEP/UNCHS Balkans Task Force (BTF). The Kosovo Conflict. Consequences for the Environment & Human Settlements. Nairobi: United Nations Environment Programme, United Nations Centre for Human Settlements (Habitat), 1999. Internet: http://www.unep.org.

4 Advisory Note on current work on DU by UNEP. Genève: United Nations Environmental Program, 2001. Internet: http://balkans.unep.ch/du/missions.html, geraadpleegd 23-2-2001.

5 Harley NH, Foulkes EC, Hilborne LH, Hudson A, Anthony CR. A Review of the Scientific Literature As It Pertains to Gulf War Illnesses. Volume 7: Depleted Uranium. Santa Monica, CA: RAND, 1999; Report MR-1018/7-OSD.

6 Committee on the Health Effects Associated with Exposures During the Gulf War. Gulf War and Health: Volume 1. Depleted Uranium, Pyridostigmine Bromide, Sarin, and Vaccines. Washington, DC: National Academy Press (Institute of Medicine), 2000. Internet: http://books.nap.edu/books/030907178X/html/R1.html.

7 Toxicological Profile for Uranium. Atlanta, GA, USA: U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, 1999.

8 Derix S, Knip K. NAVO-munitie Kosovo bevatte vrijwel zeker kernafval. NRC Handelsblad 20-01-2001.

9 UNEP confirms plutonium found in DU ammunition [press release]. Genève: United Nations Environmental Program, 2001. Internet: http://balkans.unep.ch/press/press010216.html, geraadpleegd 23-2-2001.

10 Cordfunke EHP. The Chemistry of Uranium. New York: Elsevier, 1969.

11 Ontwerp-besluit stralingbescherming. Stcrt. 08-05-2000.

12 Weast RC, Astle MJ, Beyer WH, eds. CRC handbook of Chemistry and Physics. Boca Raton, FL, USA: CRC Press Inc., 1986.

13 Browne E, Firestone RB, Shirley VS. Table of Radioactive Isotopes. New York: John Wiley and Sons, 1986.

14 Snihs JO, Åkerblom G. Use of Depleted Uranium in Military Conflicts and Possible Impact on Health and Environment. SSInews 2000; 6(December): 1-8.

15 Hedaya MA, Birkenfeld HP, Kathren RL. A sensitive method for the determination of uranium in biological samples utilizing kinetic phosphorescence analysis (KPA). J Pharm Biomed Anal 1997; 15(8): 1157-1165.

16 Ejnik JW, Carmichael AJ, Hamilton MM, McDiarmid M, Squibb K, Boyd P et al. Determination of the isotopic composition of uranium in urine by inductively coupled plasma mass spectrometry. Health Phys 2000; 78(2): 143-146.

17 Dang HS, Pullat VR, Pillai KC. Determining the normal concentration of uranium in urine and application of the data to its biokinetics. Health Phys 1992; 62(6): 562-566.

18 Hurtgen C. Natural radioactivity in bioassay by alpha spectrometry measurements [accepted for publication]. J Radioanal Nucl Chem 2001; 248(2).

19 Enquêtecommissie Vliegramp Bijlmermeer. Een beladen vlucht. Eindrapport Bijlmer Enquête. Den Haag: SDU Uitgevers, 1999.

20 Uijt de Haag PA, Smetsers RC, Witlox HW, Krus HW, Eisenga AH. Evaluating the risk from depleted uranium after the Boeing 747-258F crash in Amsterdam, 1992. J Hazard Mater 2000; 76(1): 39-58.

21 United Nations Scientific Committee on the Effects of Atomic Radiation. Report of The United Nations Scientific Committee on the Effects of Atomic Radiation. New York: United Nations, 1977; Official record of the General Assembly, 32nd Session, Supplement 40.

22 United Nations Scientific Committee on the Effects of Atomic Radiation. Sources and Effects of Ionizing Radiation 1988 UNSCEAR Report to the General Assembly, with annexes. New York: United Nations, 1988.

23 United Nations Scientific Committee on the Effects of Atomic Radiation. Sources and Effects of Ionizing Radiation 1993 UNSCEAR Report to the General Assembly, with annexes. New York: United Nations, 1993.

24 United Nations Scientific Committee on the Effects of Atomic Radiation. Sources and Effects of Ionizing Radiation. Report to the General Assembly, with Scientific Annexes. Volume I: Sources. Volume II: Effects. Annex B: Exposure for Natural Sources. New York: United Nations, 2000.

25 Smetsers RCGM, Blaauboer RO. Variations in outdoor radiation levels in the Netherlands. Bilthoven: Rijksinstitituut voor Volksgezondheid en Milieu, 1996; RIVM Rapport 610064002. Internet: http://www.rivm.nl/.

26 Köster HW, Keen A, Pennders RMJ, Bannink DW, de Winkel JH. Linear Regression Models for the Natural Radioactivity (U-238, Th-232, and K-40) in Dutch Soils. A Key to Anomalies. Rad Prot Dos 1988; 24(1-4): 63-68.

27 van de Plassche E, van de Hoop M, Posthumus R, Crommentuijn T. Risk limits for boron, silver, titanium, tellurium, uranium and organosilicon compounds in the framework of EU Directive 76/464/EEC [Risicogrenzen voor boor, zilver, titanium, tellurium, uranium en organische siliciumverbindingen in het kader van EU Richtlijn 76/464/EEC]. Bilthoven: Rijksinstitituut voor Volksgezondheid en Milieu, 1999; RIVM Rapport 601501005. Internet: http://www.rivm.nl/.

28 National Council on Radiation Protection and Measurements. Exposure of the Population in the United States and Canada from Natural Background Radiation. Bethesda, Md: NCRP, 1999; NCRP Report No. 94.

29 Golchert NW, Duffy TL, Sedlet J. Environmental Monitoring at Argonne National Laboratory. Argonne, USA: Argonne National Laboratory, 1985; Report ANL-85-17.

30 Fisenne IM, Perry PM, Decker KM, Keller HW. The daily intake of 234,235,238U, 228,230,232Th and 226,228Ra by New York City residents. Health Phys 1987; 53(4): 357-363.

31 United States Nuclear Regulatory Commission. Assessment of the public health impact from the accidental release of UF6 at the Sequoyah Fuels Corporation Facility at Gore, Oklahoma. Vol. 1. Washington DC: US NRC, 1986; Report NUREG-1189, Doc No. 40-8027.

32 Drury JS, Baldauf MF, Daniel EW, Fore CS, Uziel MS. Radioactivity in Food Crops. Oak Ridge, TN, USA: Oak Ridge National Laboratory, 1983; Doc. ORNL-5963.

33 Glastra P, Köster HW, Mattern FCM. Variations in outdoor radiation levels in the Netherlands. Bilthoven: Rijksinstitituut voor Volksgezondheid en Milieu, 1988; RIVM Rapport 248604001.

34 Glastra P, van Zanten R, Drost RMS, Mattern FCM. Onderzoek naar de radioactiviteit van drinkwater over de periode 1981-1984. Bilthoven: Rijksinstitituut voor Volksgezondheid en Milieu, 1989; RIVM Rapport 248201001.

35 Cothern CR, Lappenbusch WL. Occurrence of uranium in drinking water in the U.S. Health Phys 1983; 45(1): 89-99.

36 International Commission on Radiological Protection. Report of the Task Group on Reference Man. Oxford, UK: Pergamon Press, 1975; ICRP Publication 23.

37 Fisenne IM, Perry PM, Harley NH. Uranium in Humans. Rad Prot Dos 1988; 24(1-4): 127-131.

38 Spencer H, Osis D, Fisenne IM, Perry PM, Harley NH. Measured intake and excretion patterns of naturally occurring 234U, 238U, and calcium in humans. Radiat Res 1990; 124(1): 90-95.

39 Health and environmental consequences of depleted uranium use in the US army: Technical report. Atlanta, GA, USA: Army Environmental Policy Institute, 1995. Internet:

http://www.aepi.army.mil/library/aepi%20publications/du/techreport.html [05-01-2001 08:49:33], geraadpleegd 5-1-2001.

40 Gulf War Exposure Assessment, draft report. Aberdeen, MD, USA: Center for Health Promotion and Preventive Medicine, 1998.

41 The opinion of the group of experts established according to Article 31 of the Euratom Treaty. Depleted uranium. Brussels: European Commission, 2001.

42 International Commission on Radiological Protection. Human Respiratory Tract Model for Radiological Protection. ICRP Publication 66. Annals of the ICRP 1994; 24(1-3).

43 International Commission on Radiological Protection. Age Dependent Doses to Members of the Public from Intake of Radionuclides: Part 3. Ingestion Dose Coefficients. ICRP Publication 69. Annals of the ICRP 1995; 25(1).

44 International Commission on Radiological Protection. Age Dependent Doses to Members of the Public from Intake of Radionuclides: Part 4. Inhalation Dose Coefficients. ICRP Publication 71. Annals of the ICRP 1995; 25(3-4).

45 Leggett RW, Harrison JD. Fractional absorption of ingested uranium in humans. Health Phys 1995; 68(4): 484-498.

46 Wrenn MW, Durbin PW, Howard B, Lipsztein J, Rondo J, Still ET. Metabolism of ingested U and Ra. Health Phys 1985; 48: 601-633.

47 Eve IS. A review of the physiology of the gastrointestinal tract in relation to radiation doses from radioactive materials. Health Phys 1966; 12(2): 131-161.

48 Fisenne IM. Uranium. In: Seiler H, Sigel A, Sigels H, editors. Handbook on Metals in Clinical and Analytical Chemistry. New York: Marcel Dekker Inc., 1994: 639-649.

49 Berlin M, Rudell B. Uranium. In: Friberg L, Nordberg G, Vouk V, editors. Handbook on the Toxicology of Metals. Amsterdam: Elsevier Science Publishers, 1986.

50 Venugopal B, Luckey TD. Chemical toxicity of metals and metalloids. In: Venugopal B, Luckey TD, editors. Metal toxicity in mammals. New York: Plenum Press, 1977.

51 Priest ND, Howells GR, Green D, Haines JW. Uranium in bone: metabolic and autoradiographic studies in the rat. Hum Toxicol 1982; 1(2): 97-114.

52 Ribera D, Labrot F, Tisnerat G, Narbonne JF. Uranium in the environment: occurrence, transfer, and biological effects. Rev Environ Contam Toxicol 1996; 146: 53-89.

53 Leggett RW. Basis for the ICRP's age-specific biokinetic model for uranium. Health Phys 1994; 67(6): 589-610.

54 Ellender M, Haines JW, Harrison JD. The distribution and retention of plutonium, americium and uranium in CBA/H mice. Hum Exp Toxicol 1995; 14(1): 38-48.

55 La Touche YD, Willis DL, Dawydiak OI. Absorption and biokinetics of U in rats following an oral administration of uranyl nitrate solution. Health Phys 1987; 53(2): 147-162.

56 Tracy BL, Quinn JM, Lahey J, Gilman AP, Mancuso K, Yagminas AP et al. Absorption and retention of uranium from drinking water by rats and rabbits. Health Phys 1992; 62(1): 65-73.

57 Kathren RL, McInroy JF, Moore RH, Dietert SE. Uranium in the tissues of an occupationally exposed individual. Health Phys 1989; 57(1): 17-21.

58 Lippman M, Ong DY, Harris WB. The significance of Uranium Excretion Data. Ind Hyg J 1964;(January-February): 43-54.

59 Eisenbud MA, Quigley JA. Industrial Hygiene of Uranium Processing. Arch Indust Health 1956; 14: 12-22.

60 Hooper FJ, Squibb KS, Siegel EL, McPhaul K, Keogh JP. Elevated urine uranium excretion by soldiers with retained uranium shrapnel. Health Phys 1999; 77(5): 512-519.

61 Leggett RW. The behavior and chemical toxicity of U in the kidney: a reassessment. Health Phys 1989; 57(3): 365-383.

62 Hursh JB, Spoor NL. Uranium, Plutonium, Transplutonic Elements. New York: Springer Verlag, 1973.

63 Stannard JN. Radioactivity and Health: A History. Richland, WA, USA: Battelle Memorial Institute, 1989.

64 Dupree EA, Watkins JP, Ingle JN, Wallace PW, West CM, Tankersley WG. Uranium dust exposure and lung cancer risk in four uranium processing operations. Epidemiology 1995; 6(4): 370-375.

65 Ritz B. Radiation exposure and cancer mortality in uranium processing workers. Epidemiology 1999; 10(5): 531-538.

66 Cragle DL, McLain RW, Qualters JR, Hickey JL, Wilkinson GS, Tankersley WG et al. Mortality among workers at a nuclear fuels production facility. Am J Ind Med 1988; 14(4): 379-401.

67 Waxweiler RJ, Archer VE, Roscoe RJ, e.a. Mortality patterns among a retrospective cohort of uranium mill workers. Health Physics Society, 1983; Prtoceedings of Epidemiology Applied to Health Physics.

68 Archer VE, Wagoner JK, Lundin FE, Jr. Cancer mortality among uranium mill workers. J Occup Med 1973; 15(1): 11-14.

69 Gartland KP, Anthony ML, Beddell CR, Lindon JC, Nicholson JK. Proton NMR studies on the effects of uranyl nitrate on the biochemical composition of rat urine and plasma. J Pharm Biomed Anal 1990; 8(8-12): 951-954.

70 Leach LJ, Gelein RM, Banner BJ, e.a. The Acute Toxicity of the Hydrolysis Products of Uranium Hexafluoride (UF6) When Inhaled by Rat and Guinea Pig, final report. 1984; Report ISS K/SUB-81-9039-3, NTIS DE84011539.

71 Mao Y, Desmeules M, Schaubel D, Berube D, Dyck R, Brule D et al. Inorganic components of drinking water and microalbuminuria. Environ Res 1995; 71(2): 135-140.

72 Kathren RL. The United States Transuranium and Uranium Registries 1968-1993. Rad Prot Dos 1995; 4: 349-354.

73 Zamora ML, Tracy BL, Zielinski JM, Meyerhof DP, Moss MA. Chronic ingestion of uranium in drinking water: a study of kidney bioeffects in humans. Toxicol Sci 1998; 43(1): 68-77.

74 Hotz P, Buchet JP, Bernard A, Lison D, Lauwerys R. Renal effects of low environmental cadmium exposure: 5year follow-up of a subcohort from the Cadmibel study. Lancet 1999; 354: 1508-1513.

75 Domingo JL, Llobet JM, Tomas JM, Corbella J. Acute toxicity of uranium in rats and mice. Bull Environ Contam Toxicol 1987; 39(1): 168-174.

76 Pavlakis N, Pollock CA, McLean G, Bartrop R. Deliberate overdose of uranium: toxicity and treatment. Nephron 1996; 72(2): 313-317.

77 Vich Z, Kriklava J. Erythrocytes of uranium miners: the red blood picture. Br J Ind Med 1970; 27(1): 83-85.

78 Kathren RL, Moore RH. Acute accidental inhalation of U: a 38-year follow-up. Health Phys 1986; 51(5): 609-619.

79 Butterworth A. The significance and Value of Uranium in Urine Analysis. Trans Assoc Ind Med Offrs 1955; 5: 36-43.

80 Maynard EA, Down WL, Hodge HC. Oral Toxicity of Uranium Compounds. In: Voegtlin C, Hodge HC, editors. Pharmacology and Toxicology of Uranium Compounds. New York: McGraw-Hill, 1953.

81 McDiarmid MA, Keogh JP, Hooper FJ, McPhaul K, Squibb K, Kane R et al. Health effects of depleted uranium on exposed Gulf War veterans. Environ Res 2000; 82(2): 168-180.

82 Gezondheidsraad: Commissie Stralingsrisico's. Stralingsrisico's. Evaluatie van de wetenschappelijke gegevens over de gezondheidsrisico's van blootstelling aan ioniserende straling. Den Haag: Gezondheidsraad, 1991; Publicatie nr 1991/22.

83 International Commission on Radiological Protection. 1990 recommendations of the International Commission on Radiological Protection. ICRP Publication 60. Annals of the ICRP 1991; 21(1-3).

84 Richtlijn 96/29/Euratom van de Raad van 13 mei 1996 tot vaststelling van de basisnormen voor de bescherming van de gezondheid der bevolking en der werkers tegen de aan ioniserende straling verbonden gevaren. Publikatieblad 1996; L159(29/06/1996): 1-114.

85 American Conference of Govermental Industrial Hygienists. Threshold Limiting Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Cincinnati, OH, USA: ACGIH, 2000.

86 Ministerie van Sociale Zaken en Werkgelegenheid. Nationale MAC-lijst 2000. Den Haag: Sdu Uitgevers, 1999.

87 Lauwereys RR, Hoet P. Uranium. Industrial Chemical Exposure: Guidelines for Biological Monitoring. Boca Raton, FL, USA: Lewis Publishers, 1993.

88 Gezondheidsraad. Bezorgdheid over lokale milieufactoren. Den Haag: Gezondheidsraad, 2001; Publicatie nr 2001/10.

Abbreviations and terms

1 Abbreviations

ACGIH	American Conference of Governmental Industrial Hygienists
AED	Aerodynamic equivalent diameter
ALARA	As Low As Reasonably Achievable
BTF	Balkan Task Force
DU	Depleted Uranium
ICRP	International Commission on Radiological Protection
KFOR	Kosovo Force
NAVO	Noord-Atlantische Verdrags Organisatie
RAND	National Defense Research Institute
RIVM	Rijksinstituut voor Volksgezondheid en Milieu
TLV	Threshold Limit Value
TWA	Time-Weighted Average
UNEP	United Nations Environment Programme
UNCHS/Habitat	United Nations Center for Human Settlements
UNSCEAR	United Nations Scientific Committee on the Effects of Atomic Radiation

2 Glossary of radiological terms

Activity	The number of disintegrations per second in a quantity of radioactive material (such as DU). The unit of activity is the becquerel (Bq).
Becquerel (Bq)	A unit of activity. 1 becquerel (Bq) equals 1 disintegration (instance of radioactive decay) per second (1 Bq = 1 s^{-1}).
Contamination	Contamination of a material, a surface, an environment or a person by radioactive material (such as DU). A person is said to be internally contaminated if he or she absorbs the radioactive material into his or her body.
Exposed worker	A person who, in the course of his or her work, is exposed to radiation which eventually may cause him or her to receive a radiation dose in excess of the dose limit for members of the general public.
Exposure	Being exposed to ionising radiation. Distinction is made between external exposure, where the radiation source is outside the body, and internal exposure, where a radioactive substance is absorbed into the body.
Deterministic effect	An effect of radiation that always occurs if the radiation dose is in excess of a given level (in contrast to a stochastic effect).
(Committed) effective dose	Sum of the (committed) equivalent organ doses resulting from the absorption of uranium, each multiplied by the appropriate organ weighting factor. The unit of committed effective dose is the sievert (Sv).
Committed equivalent dose	The time integral of the equivalent dose rate in an organ, as received by an individual as a result of the absorption of a radioactive substance into the body. Where adults are concerned, the period used is fifty years. The unit of committed equivalent dose is the sievert (Sv).
Absorbed organ dose	The amount of energy absorbed per unit mass, averaged across a given organ. The unit of absorbed dose is the gray (Gy).
Gray (Gy)	A unit of absorbed dose. 1 gray equals 1 joule per kilogram (1 Gy = 1 $J.kg^{-1}$).
Isotopes	Nuclides of a single element, which have the same number of protons, but a different number of neutrons. Examples include U-234, U-235 and U-238 (isotopes of the element uranium).
Sievert (Sv)	A unit of equivalent or effective dose. 1 sievert equals 1 joule per kilogram (1 Sv = 1 $J.kg^{-1}$).
Stochastic effect	An effect of radiation, the likelihood of which depends on the radiation dose received. It is assumed that there is no threshold dose for a stochastic effect.
Organ weighting factor (w_T)	Non-dimensional factor used to weight the equivalent dose to an organ (T). The factors for the various organs were proposed by the ICRP[83] and are incorporated into European[84] and Dutch[11] regulations.

Annexes

Annex **A**

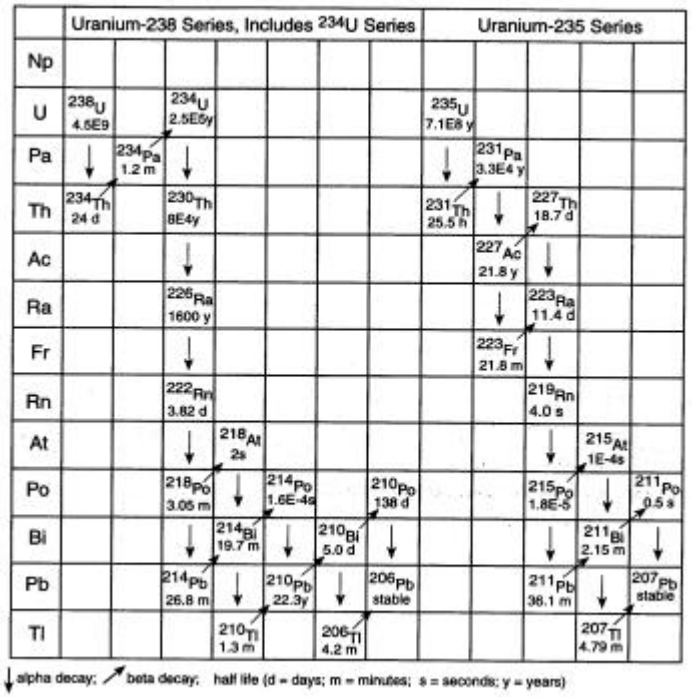
The committee

- dr WF Passchier, *chair*,
  Dep. Executive Director, Health Council of the Netherlands, The Hague
  and Professor of Risk Analysis, Department of Health Risk Analysis and
  Toxicology, Universiteit Maastricht, the Netherlands
- dr ME de Broe
  Professor of Nephrology, University of Antwerp,Wilrijk, Belgium
- dr C Hurtgen
  Radiochemist; Study centre of Nuclear Energie, Mol, Belgium
- dr AS Keverling Buisman
  Radiation Protection Supervisor; Netherlands Energy Research Foundation,
  Petten, The Netherlands
- dr J Noordhoek
  Professor of Toxicology; University of Nijmegen, Nijmegen, The Netherlands
- dr MM Verberk
  Epidemiologist, toxicologist, Academic Medical Center, University of
  Amsterdam, The Netherlands
- dr NPE Vermeulen
  Professor Molecular toxicology, Vrije Universiteit Amsterdam, The Netherlands
- ir JWN Tuyn, *secretary*
  Health physicist, The Hague,The Netherlands

Annex **B**

Decay schemes of uranium-238 and uranium-235

Decay schemes of uranium-238 (including uranium-234) and of uranium-235. Each step shows the radionuclide concerned and its radiological half-life. A downward arrow indicates transition involving the emission of an alpha particle; a tilted upward arrow indicates transition involving the emission of a beta particle.



Annex **C**

Exposure scenarios and the associated radiation doses

In order to estimate the risks of exposure to DU, it is necessary to develop exposure scenarios. Both UNEP's Balkan Task Force[3] and a team of Euratom experts[41] have designed such scenarios. To some extent, these scenarios draw upon earlier work carried out by the Swiss consultancy Brenk Systemplanung (BS).

Scenario	Consequences/Comments
Common assumptions of all scenarios ▪ 10 kg DU ▪ Particles less than 10 µm and barely soluble (ICRP class S) ▪ Contaminated surface: 1000 m² ▪ Calculation of effective dose for adults	In accordance with scenario described by UNEP. Brenk Systemplanung evaluates implications over a period of 1000 years.
Fragments of DU picked up This scenario assumes that fragments of DU are picked up and (a) saved in a bag, (b) worn as decoration, (c) placed next to a bed.	▪ Skin dose is low after first few weeks (0.6 µSv/year) ▪ Skin dose after 500 hours may be high, but not sufficient to induce deterministic effects ▪ Skin dose 0.6 µSv/year.
Solid pieces of DU in or on the ground Involves external exposure only	As *Fragments of DU picked up* or *External radiation.*
Inhalation of resuspended dust This scenario assumes that people inhale air in which DU particles are suspended. The dust originates from an explosion and has settled over an area of 1000 m² around the detonation site, before being resuspended by the wind.	The dust contains 6 µg of DU per mg of material. This leads to the inhalation of 0.6 to 60 µg DU. The effective dose is low (0.07 to 7 µSv).

All DU dust is contained in the top 1 mm of soil. The particle concentration in the air is between 50 and 5000 µg/m³. The residence time is two hours.	
Ingestion of DU	
Soil in mouth A child plays in a contaminated area and puts soil in its mouth. DU concentration is 6 µg per mg of soil. The child swallows 1 g of soil.	Effective dose is low (4 µSv), but possibly sufficient to induce acute chemical toxic effects.
Contaminated vegetables A person eats vegetables grown in the contaminated area, before rain has rinsed off the DU. Surface contamination before rainfall is 10 g/m². Vegetable intake is 60 kg per year or 1 kg per week. The vegetables are taken from 1 m² of ground.	Intake is approximately 100 mg of DU. This is significant on account of DU's chemical toxicity. The effective radiation dose is approximately 0.1 mSv. Washing would reduce contamination by 99 per cent.
Contaminated hands A person's hands are contaminated with soil containing DU in the concentration described in *Soil in mouth* scenario. The amount of DU swallowed is 10 to 100 times less than in the latter scenario.	Effective dose is low (0.04 to 0.4 µSv).
Contaminated groundwater Groundwater is contaminated as DU scattered over the surface dissolves. Water is subsequently used as drinking water. Groundwater is 3 m below ground level. 10 per cent of the DU at the surface disappears per year. The water concentration in the soil is 30 per cent.	Doses are within drinking water standards. UNEP scenario assumes no movement of groundwater, while BS assumes water is flowing. UNEP's assumption results in a more pessimistic forecast of radiation dose, i.e. approximately 1 mSv per year, while BS's assumption leads to a much lower estimate: 24 µSv per year. The BS scenario appears more plausible.
Contaminated food Food is contaminated due to livestock grazing on contaminated ground or due to vegetables being grown on ground where groundwater is contaminated.	Doses are very low if precautions taken. Meat and milk: less than 0.1 mSv per day; solution – don't let livestock graze on contaminated land UNEP scenario suggests an effective dose through ingestion of 7 µSv/year if plants that have absorbed DU through their roots are eaten
External radiation Members of the general public suffer external exposure to radiation from DU particles spread across the area. The DU is mixed with the top 10 cm of soil (70 mg DU per kg soil).	Doses received by general public very low: 4 µSv/year

Annex **D**

Dose conversion coefficients for DU

This annex gives committed equivalent doses following the intake of 1 Bq of DU. The values are taken from ICRP Publications 69 and 71.[42,44]

Dose conversion coëfficiënten for inhalation in µSv/Bq under the assumption of adults inhaling dust with an AMAD of 1 µm and of DU-compounds belonging to ICRP class S (slightly soluble)	
Bone surface	0.47
Kidneys	0.17
Red Bone Marrow	0.049
Respiratory organs: upper tracts	32
Respiratory organs: lungs	67
Dose conversion coëfficiënten for inhalation of DU in µSv/Bq under the assumption that the DU-compounds are slightly soluble.	
Red Bone Marrow	0.759
Lungs	0.0255